THE operation to correct mitral stenosis has proved its value in the past 8 years. Aortic stenosis can also be palliated surgically but the results are less dramatic, the risk is higher, and the pathologic process often limits the quality of the repair. Operations for mitral and aortic insufficiency are still in the experimental stage, but are improving. This article is concerned chiefly with the selection of patients with predominant mitral stenosis for operation. Such an operation has been termed by us “valvuloplasty.” Operations with similar objectives, i.e., fracturing and cutting fused commissures, separating fusion of the chordae tendineae, and withall returning the maximum valvular action to the stenotic complex have been variously designated as “commissurotomy,” “valvulotomy,” or “valvotomy.”

Physicians treating patients with rheumatic mitral valve disease are now faced with the question whether or not to recommend operation. In making such a decision several factors are involved. As in any condition where there are alternative medical and surgical treatments, the decision requires knowledge of the specific medical prognosis, the operative risk, and the results to be gained from surgery.

Mitrail Stenosis

Summary of Present Indications for Operation in Predominant Mitral Stenosis

Our opinion as to which patients should be offered operation has not changed since our early experience. The results of surgery have borne out our original concepts and these have now been generally accepted.1-4 Patients with the murmur of mitral stenosis but without symptoms (group I) do not need operation. Many of them remain asymptomatic for many years. It must be remembered that the operation for mitral stenosis is a good palliative but not a curative operation, although the palliation is often life-saving. In patients with moderate but nonprogressive symptoms (group II), the operation may be considered elective. The decision depends on the patient's social, economic, and psychologic situation. The patients in this as well as in other groups should be observed with care and given medical treatment when possible before advising surgery. Frequently, with simple medical measures and readjustment of the patient’s life the symptoms become remarkably ameliorated. Some unusual situation such as pregnancy or severe infection may have precipitated cardiac symptoms. Without such stress the patient may enjoy a good state of compensation.

Patients in group III, disabled by progressive symptoms, chiefly dyspnea, but who have not yet become complete cardiac invalids or progressed to chronic persistent congestive failure, need the operation urgently. The risk at the present time is very low and the results are excellent. The medical prognosis of these people is poor.

Group IV patients are cardiac invalids condemned to a life of complete inactivity under medical treatment, with a relatively short life expectancy. Although the operative risk in these patients remains high, the results are surprisingly good and operation should be offered. Physicians should be alert, however, to urge operation while these patients are still in group III when much can be accomplished at a low operative risk. It is bad practice to wait
until they have slipped into group IV. While the results of surgery contrast brilliantly with medical therapy in this category, we must not let this conceal the great disservice that has been done the patient by allowing him to move from group III into IV. The operative risk to the patient has increased perhaps 30 times and his potentialities for rehabilitation have diminished.

Operative and Postoperative Prognosis of Mitral Stenosis

Our conclusions as to the value of the procedure are based on a study of 1,000 consecutive patients operated upon for predominant mitral stenosis by one of us (D. E. H.).

Many of the patients in this group were diagnosed preoperatively as having some degree of mitral insufficiency in addition to the stenosis. Patients with minor degrees of aortic valve disease and with tricuspid incompetence were included. We have previously reported a follow-up study of the first 500 of these patients1 and the more recent data bear out the conclusions reached at that time.

No patients in group I have been operated upon. There have been only 19 in group II, and these have been included in group III for statistical purposes. Group III patients constituted nearly three fourths of the entire group and the remainder were group IV patients.

The mortality of mitral valvuloplasty in the first consecutive 1,000 has steadily fallen, so that the risk in group II and group III patients is now less than 1 per cent. There were but 2 deaths among the group III patients in the last 500 of this series. However, the group IV mortality remains high at about 20 per cent. This emphasizes the importance of operating on patients before they slip from group III to group IV. This deterioration can usually be anticipated.

Seventy-eight per cent of the patients in group III and 62 per cent of patients in group IV showed significant postoperative improvement at the time of the most recent follow-up report. Criteria for improvement have been defined elsewhere.1 The degree of improvement in group III patients has been maintained over a follow-up time up to 5 years (fig. 1). Thus the improvement from operation is persistent, and the number of significant regressions is small. The same is true for group IV patients. The degree of improvement in group III patients after 6 or more years is less than for the first years but the differences are not significant because of the small number of patients followed.

Some skeptics argue that improvement may be due to the psychologic effect of the procedure or to better medical care and not to the operation itself. We have data, as have others, indicating that the hemodynamics of the circulation actually improve after the operation in a considerable number of patients. This long-term clinical study of a large number of patients constitutes further evidence. The simple fact that the good results persist suggests that the operation has fundamentally altered the patient's course. The ultimate test of the value of this operation, of course, will be whether these patients will survive longer than comparable patients treated medically.

Prognosis of Patients with Mitral Stenosis Treated Medically

It is almost impossible to obtain data on medically treated patients similar to this surgical series of 1,000. Most studies are either statistically invalid or the groups not easily comparable. Some studies based on autopsy statistics are retrospective, so that it is almost impossible to determine when and to what degree the patients became symptomatic. Grant4 reported a group of soldiers with rheumatic heart disease followed 10 years after World War I. This important study dealt with young males only. He found that 45 per cent of
the patients who originally had poor exercise
tolerance were dead at the end of 10 years, and
all were dead who had shown congestive
symptoms.

A study by Wilson and Greenwood
on life expectancy, which is not strictly comparable
with ours, indicates that about 50 per cent of
their patients who originally had shown severe
pulmonary congestion or atrial fibrillation were
dead in 5 years, and 60 per cent of those who at
any time had shown right heart failure were
dead at this point. Hamilton and Thomson
ited statistics on women in the child-bearing
age to the effect that 20 per cent of the “favor-
able” and 63 per cent of the “unfavorable”
cardiac patients were dead after 10 years. Most
of their “favorable” patients would belong in
class 1 and 2 in the American Heart Association
or our classifications. Most of the “unfavor-
able” group would fall in our groups III and IV.

The only series comparable to ours of which
we are aware is that by Olesen, who studied a
group of medically treated patients first ob-
served between the years of 1933 and 1949 in
Copenhagen. One hundred seventy-six of these
patients were classified according to the Amer-
ican Heart Association in class 3 or were
fibrillating patients in class 2, and 52 were in
class 4. These are comparable to our groups III
and IV. The average age and the sex distribu-
tions are almost exactly the same as in our
patients. What then are the results in our series
of operated patients as compared with Olesen’s
medically treated patients, so far as survival is
concerned? Figure 2 shows the results in group
III patients over a 7-year period. Of the medi-
cally treated patients, 51 per cent survived for
7 years. Of the surgically treated patients the
survival rate was 87 per cent after 7 years,
including an operative mortality of 3 per cent.
Thus the survival rate in the operated patients
was significantly better than in Olesen’s group
of medically treated patients. One must bear in
mind, of course, that many of these medically
treated patients were observed prior to some of
the more recent advances in cardiac thera-
petus and, in particular, before the advent of
antibiotic treatment.

The group IV patients have been similarly
compared and gave even more striking results
(fig. 3). In our group of operated patients
survival rate has been 54 per cent at the end of
7 years, including the mitral operative mort-
ality of 24 per cent. Only 6 per cent of Olesen’s
medically treated patients were alive at the
end of 6 years. In comparing the 2 groups, the
operated patients are still better off as far as
survival is concerned even at the end of the
first year of observation, which includes the
operative mortality. The difference between the
survival of operated and medically treated pa-
tients is so striking in both groups III and IV
that it is evident that the operation for mitral
stenosis has increased life expectancy. More-
ever, the operated patients have, for the most
part, lived lives of much greater comfort and
usefulness, in contrast to the deterioration that
characterizes the patients under medical
treatment.

Preoperative Evaluation of the Patient with
Mitral Valvular Disease

The preceding discussion has been concerned
with over-all results of a large group of patients
who were diagnosed preoperatively as having
predominant mitral stenosis. When individual patients are under consideration for operation, the degree of mitral stenosis and the extent to which the symptoms are attributable to obstruction at the mitral valve or to other factors must be assessed.

**Degree of Stenosis**

Insistence that symptoms be present and either disabling or progressive before considering mitral valvuloplasty has proved to be a good method of limiting surgery to patients with hemodynamically significant stenosis. It allows those with only "auscultatory" stenosis to avoid surgery. Although an occasional asymptomatic patient with severe stenosis changes to a rapid deteriorating course, the symptomatic stage of mitral stenosis is generally of gradual onset and there is ample clinical warning before life is threatened and operative risk is significantly increased.

There are patients whose symptoms are atypical or whose history is difficult to interpret. These include patients who tend to minimize symptoms and conceal disability. They often adjust their lives remarkably to their limited capacity. Conversely, there are patients unusually aware of symptoms. This group includes patients with cardiac neurosis or neurocirculatory asthenia. Careful history from family and friends and prolonged observation of the patient may help in clinical assessment. Right ventricular hypertrophy by electrocardiogram or enlargement of the right ventricle and pulmonary artery by fluoroscopy suggests significant stenosis. Often a valid clinical decision can only be made by means of objective tests of cardiac function, e.g., cardiac catheterization, or over-all cardiopulmonary performance, as suggested by Bruce. If the lesion is pure mitral stenosis and no other cardiac or pulmonary disease is likely, direct evidence of pulmonary hypertension and elevated pulmonary wedge ("capillary") pressure, especially during exercise, constitute adequate indications for operation. This is one group of patients in which catheterization of the right heart may still be of clinical value.

When, however, the clinical signs of mitral stenosis are equivocal, and particularly when there is also suspicion of mitral insufficiency, aortic valve disease, or myocardial failure, right heart catheterization is not likely to be of diagnostic help, and the method of choice is percutaneous catheterization of the left heart. This was first described by Björk and associates in Sweden in 1953. The technic was modified and improved by Fisher in 1954. With the patient in the prone position a needle is inserted under fluoroscopic guidance through the skin of the back into the left atrium. A catheter is then passed through the needle across the mitral valve into the left ventricle. Pressures on both sides of the mitral valve are measured successively or preferably simultaneously (fig. 4 bottom). The mean pressure difference (gradient) across the mitral valve during diastole is thus obtained. There is no pressure difference across the normal mitral valve during diastole (fig. 4 top). Narrowing of the mitral valve constitutes an obstruction in the face of which diastolic blood flow can only be maintained by a rise in left atrial pressure producing

![FIG. 4. Catheterization of the left heart in mitral stenosis. Top. The pressure tracings obtained during withdrawal of a catheter from the left ventricle to the left atrium across a normal mitral valve demonstrate that there is no pressure difference across the valve in diastole. Bottom. Withdrawal from the left ventricle to the left atrium across a stenosed mitral valve shows a mean diastolic pressure gradient of 17 mm. Hg. Cardiac output determined by simultaneous catheterization of the right heart was 3.8 L./min. By means of the formula of Gorlin and Gorlin the diastolic mitral valve area was estimated to be 0.7 cm.² Severe mitral stenosis was found at subsequent mitral valvuloplasty (D. E. H.).](http://circ.ahajournals.org/)

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a pressure difference across the mitral valve. Blood flow is a function of both the size of the valve orifice and the pressure gradient across it in diastole. If the pressure gradient and blood flow are measured simultaneously, functional valve size may be estimated from hydraulic formulas. These were developed by Gorlin and Gorlin\textsuperscript{13} at a time when the pressure gradient could not be measured directly but only assessed as the difference between the pulmonary wedge pressure determined by right heart catheterization and an assumed left ventricular diastolic pressure.

Measurement of the pressure gradient across the mitral valve by percutaneous left heart catheterization or at the time of thoracotomy will give information as to presence or absence of obstruction, and may allow some general judgment as to its severity if the arterial blood pressure is normal. Quantification of stenosis, however, demands a simultaneous determination of the cardiac output. Left heart catheterization alone or in combination with right heart catheterization has been performed in our laboratories on approximately 150 occasions without serious complications. Our experience and that of others\textsuperscript{12, 14} suggest that this technic carries a low risk when performed with care in selected patients. The procedure remains to be fully developed and evaluated but it shows great promise. It is the most direct physiologic approach to the mitral valve yet devised.

The Differentiation of Stenosis from Insufficiency

Surgery of mitral valve disease has made more imperative quantitative differentiation of degrees of stenosis and insufficiency and has also aided in elucidating the diagnostic criteria. It is clear that a simple diagnostic differentiation based on the presence of apical systolic or diastolic murmurs is not only inadequate but often misleading. It is true that loud apical systolic murmurs transmitted laterally suggest the presence of mitral insufficiency but they furnish little quantitative information; insufficiency may be severe with slight murmurs or insignificant with loud murmurs. Many patients have associated tricuspid insufficiency, which may give rise to systolic murmurs difficult to distinguish from those arising at the mitral valve, although they are usually not transmitted laterally of the apex. The same applies to diastolic murmurs; marked rumbling diastolic murmurs with presystolic accentuation usually mean stenosis, but stenosis may be severe with slight diastolic murmurs or rarely none at all, and patients with free insufficiency may have loud diastolic murmurs, usually, however, rather early in timing. A sharp mitral first sound and an opening snap both point to stenosis.

Patients with predominant insufficiency frequently have less pulmonary hypertension and a relatively lower effective forward blood flow than those with stenosis who are at a comparable stage of the disease. Hence the former are somewhat more likely to complain of fatigue as a dominant symptom rather than dyspnea. However, too much reliance should not be placed on this differentiation because exceptions are frequent. The presence of pulmonary hypertension is reflected in an increased and often split pulmonic second sound, prominent pulmonary artery shadows shown roentgenographically, and right ventricular enlargement. All of these are more common in mitral stenosis, whereas left ventricular enlargement not otherwise explained is a sign pointing to mitral insufficiency. It is often very difficult to be sure from roentgenogram or fluoroscopy to what degree right and left ventricular hypertrophy contribute to the enlarged cardiac silhouette, especially if the left atrium is markedly dilated. The electrocardiogram is often of greater help in estimating the type of enlargement. Unusual enlargement of the left atrium is found most commonly in association with insufficiency, and in the oblique view the enlarged atrium is characteristically low and may rest on the diaphragm. Systolic expansion of this chamber has not proved to be a helpful sign in differentiation. Calcification of the valve usually means that at least some stenosis is present.

Methods for the detection of mitral insufficiency by injection of radiopaque dye into the left atrium or ventricle have been reported but are still to be considered in the experimental stage.
In summary, there is no absolute method of differentiating stenosis from insufficiency clinically. All of the evidence must be weighed and a decision reached after such total evaluation. As will be seen, laboratory study helps little in this problem.

When significant amounts of blood regurgitate backward across the mitral valve in systole, forward mitral valve flow during diastole must increase if aortic valve flow or cardiac output is to be maintained. This increase in systolic flow across a narrowed mitral valve will be possible only if the pressure gradient is increased by a rise in left atrial pressure. In other words, insufficiency superimposed on anatomic stenosis of a valve increases physiologic stenosis by increasing the demand for forward flow. Since there is, as yet, no dependable method of measuring valve flow when regurgitation is present, although current work in our and other laboratories shows some promise that analysis of indicator-dilution curves may help in this regard, this method of estimating valve size by means of left heart catheterization does not apply when there is valvular insufficiency. As a rule, the presence of insufficiency is signaled by a regurgitant wave inscribed on the left atrial pressure tracing. The atrial pressure tracing, however, has little quantitative value, nor does its derivative, the pulmonary wedge pressure tracing. Neither has been found helpful by us in estimating the degree of mitral insufficiency or of stenosis. Nevertheless, analysis of all clinical and physiologic data in a given patient usually permits a correct decision.

Predominant mitral insufficiency constitutes a contraindication to the conventional operation for stenosis. On the other hand, when stenosis predominates and is at least moderate in degree, surgical treatment of the stenosis alone will be followed by worth-while improvement in more than half of the patients even in the presence of insufficiency. In the past 3 years mild to moderate degrees of associated regurgitation seem to have been improved by a simple extracardiac maneuver that involves the insertion of an external baffle that tends to reduce the valvular and atrial herniation and distorts the annulus so that the leaflets approximate better. Definitive correction of regurgitation has been unsatisfactory to date.

Peripheral Embolization

This is one of the great hazards to patients with mitral stenosis, particularly if they are fibrillating. No satisfactory medical preventive treatment has yet evolved. Our experience in this regard is as follows: in 19 per cent of the first 1,000 operated patients one or more clearcut episodes of peripheral embolization had occurred prior to operation. The present risk of developing an operative embolus in patients is 2 per cent in group III and 9 per cent in group IV, but it is twice as great in patients with a history of preoperative embolus. Once the hurdle of operation is safely passed, however, the likelihood of a late embolus appears to be slight. Of the entire group of 913 patients surviving operation 19 have developed late peripheral emboli. Since the average time of follow-up in the series is nearly 3 years, this represents an experience of more than 2,500 patient years or a rate of 0.6 per cent per year. This figure indicates that operation protects against late embolization. This presumably is due to reducing stasis in the atrium by valvuloplasty and removing a nidus of extreme stagnation by concomitant appendectomy. Operation may be recommended as a proper procedure in patients who have had previous emboli. It is probably more reliable and carries less risk than other radical treatments in the prevention of emboli in such patients.

How soon operation should be undertaken after a major peripheral embolism is uncertain. We have had considerable experience with patients operated upon hours, days, and weeks after peripheral embolization. The incidence of operative embolization is no greater than when the operation is delayed. Moreover, even after such a recent embolic phenomenon, thrombi in the atrium and atrial appendage are found at operation in only about half of the cases. Probably the ideal approach is to keep a patient on anticoagulant therapy for 3 or 4 weeks after a peripheral embolus and then operate as soon as possible after the omission of anticoagulant therapy permits the clotting mechanism to re-
turn to normal. Occasionally, the hand of the surgeon is forced in patients who are having showers of peripheral emboli. In such instances mitral valvuloplasty may actually become an emergency procedure.

**Rheumatic Activity**

Rheumatic activity with myocardial failure as opposed to valvular obstruction causing symptoms is a recurring problem. There is no good test for rheumatic activity. Most patients do not show the classical criteria of rheumatic fever. There is no correlation between Aschoff nodules in the auricular biopsies and the clinical course of these patients. Various laboratory tests useful in young people with active rheumatic fever are of little help in these patients, who are usually adults with subclinical rheumatic activity. Antistreptolysin titters and determinations of C-reactive protein usually are normal and sedimentation rates are not likely to be useful. Many patients with high-grade mitral stenosis, particularly those having some degree of congestive failure, show slightly elevated sedimentation rates even in the absence of any evidence of rheumatic fever. If flagrant rheumatic activity is obvious, operation should be postponed. If doubt exists as to rheumatic activity and there is clear evidence of symptoms due to obstruction, operation is usually worthwhile. This removes one cardiac burden and the patient may handle his rheumatic carditis better.

**Myocardial Failure**

It is often difficult if not impossible to determine clinically to what degree symptoms of pulmonary or systemic congestion are secondary to severe obstruction to blood flow or to myocardial insufficiency. Courmand and his group recently stressed the need for further study of this problem. A clinical course characterized by few symptoms and little disability, interrupted periodically by acute episodes of symptoms of congestion, especially in the elderly patient, suggests that there is episodic failure of the myocardium. An unusually large heart, not explained on the basis of coexistent mitral insufficiency or aortic disease, suggests poor ventricular muscle. Right heart catheterization may give support to the suspicion of prominent myocardial disease when pulmonary hypertension is absent or mild and when the arteriovenous oxygen difference is very wide, (i.e., the cardiac output very low). Since the left ventricular diastolic pressure can be measured by left-sided cardiac catheterization, a direct method of testing left ventricular competence is now available. To what extent it will be possible by such techniques to apportion symptoms between valvular lesions and myocardial weakness remains to be seen, but the approach promises to be fruitful.

**Associated Aortic Valvular Disease**

Evidence of aortic valve deformity without significant physiologic effect does not alter the risk and prognosis of mitral valvuloplasty. Significant aortic stenosis requires an elevated left ventricular systolic pressure and increased left ventricular work. In such a situation correction of mitral stenosis alone is improper as the operative risk is increased, the benefit to be expected is uncertain, and the danger of significant postoperative mitral insufficiency quite real. The clinical problem lies in the interpretation of the signs of aortic stenosis. This will be discussed later. Significant aortic stenosis per se indicates direct surgical attack just as mitral stenosis does. If both are significant, they can be corrected simultaneously. Significant aortic insufficiency, generally manifested by a lowered diastolic blood pressure and a dilated left ventricle with increased amplitude of pulsations, contraindicates mitral valvuloplasty, but does not commonly accompany pure severe mitral stenosis. The diastolic murmur heard so frequently along the left sternal border in patients with mitral stenosis is often due to pulmonic rather than aortic insufficiency.

**Associated Tricuspid Valvular Disease**

Functional tricuspid insufficiency, especially in association with atrial fibrillation, is quite frequent in patients with mitral stenosis. Organic tricuspid disease is fairly rare. Tricuspid stenosis does occur and its recognition is important since it is an operable lesion. Functional tricuspid insufficiency is no contraindication to mitral valvuloplasty. It is likely
to improve after successful correction of stenosis. Organic tricuspid insufficiency also is likely to improve after mitral valvuloplasty reduces right ventricular systolic pressure. Finally, relative tricuspid stenosis resulting from asymptomatic asymmetric commissural fusion and tight bowstring orifice occlusion by atrial enlargement may disappear after pulmonary hypertension is relieved.

Associated Pulmonary Disease

Patients with severe pulmonary vascular disease have shown marked postoperative improvement and those with the highest pulmonary arterial pressure often benefit most. Pulmonary hypertension has fallen more rapidly than the vascular changes could be expected to subside. This leads to the hypothesis that pulmonary hypertension may be in part functional.

Significant independent pulmonary disease may affect risk and prognosis. Pulmonary emphysema increases operative risk and favors residual postoperative cardiopulmonary symptoms. Pleural disease may decrease pulmonary reserve and cause early postoperative difficulties. Tests of ventilatory function and occasionally bronchospirometry may be helpful in such problems. Prominent cough and sputum, clubbing of fingers, and polycythemia are findings that should lead to a search for pulmonary disease. An approach through the right chest has been described and might be considered when there is diminished function of the right lung, and left thoracotomy could be most dangerous. Such an awkward approach, however, must be weighed against the significantly poorer quality of valvular surgery per se. Direct surgery requires the surgeon’s best effort and any handicap should be gravely considered before acceptance.

Subacute Bacterial Endocarditis

This condition like thyrotoxicosis and acute rheumatic activity should arrest the attention of the careful clinician but, once reasonably excluded, should not constitute a basis for procrastination. Prompt blood cultures should be taken. If positive, surgery is deferred until the condition is controlled.

Pregnancy

Most women with the murmur of mitral stenosis can go through pregnancy without difficulty. Patients with mild symptoms often tolerate pregnancy if carefully managed. It is seldom necessary to add the hazard of an operation at the time of pregnancy. Moreover, women with or without heart disease often retain fluid, complain of dyspnea, and present other symptoms that make it very difficult to determine the extent of actual cardiac embarrassment. Murmurs and other objective cardiac signs are frequently difficult to evaluate during pregnancy. There are, however, patients with pure mitral stenosis who would have been classified in group III or IV prior to pregnancy, patients for whom valvuloplasty would have been clearly indicated. If such patients come under observation for the first time during the first trimester of pregnancy, a mitral operation can be done with little more risk than would obtain in the nonpregnant state and probably less than for the alternative courses of action: either interruption of pregnancy and then valvuloplasty, or continuation of pregnancy under medical management and then a later valvuloplasty. From the fourth to the eighth month the added risk of valvuloplasty during pregnancy is very real and a decision as to the course of action must depend on weighing all factors. After the eighth month even patients in congestive failure should not be operated upon if it can possibly be avoided, since the burden of pregnancy becomes less during the final month.

Refusion

It has been increasingly apparent that the quality of a valvuloplasty as performed by the surgeon is of paramount importance in determining long-term results. Many of the poor results and regressions are due to inadequate operations rather than refusion of the valve. Our follow-up statistics as well as those reported by others suggest that this does not occur commonly, at least in the first 5 years. However, there is little doubt that it does occur occasionally and as the years of follow-up observation lengthen, will be seen more frequently. After
all we do not know what are the factors and mechanisms that led to the development of tight mitral stenosis in the first place, usually years after the initial rheumatic infection. Such mechanisms may still be operative in many patients after mitral valvuloplasty.

Aortic Stenosis

This lesion presents a problem of a different order. Aortic stenosis calls for compensation by left ventricular hypertrophy and patients with the disease may remain asymptomatic for many years, often into old age. By the time symptoms appear, the valve is likely to be quite fixed, calcified, and associated with myocardial fibrosis. Coronary artery disease may have developed. Symptoms usually indicate a breakdown of compensatory mechanisms and herald a rapid, worsening course. The surgical approach, whether transventricular or transaortic, is more difficult and more dangerous than the approach to the mitral valve. This disease occurs most often in males, becomes symptomatic late in its course, usually in mature or later life, and generally carries a poor medical prognosis once symptomatic. If this is taken into account, the operative mortality of about 20 to 25 per cent for the transventricular approach and 10 to 15 per cent for the newer and preferred transaortic approach does not appear prohibitive.

Diagnosis of Aortic Stenosis

A clinical diagnosis of dynamically significant aortic stenosis may be made with confidence when there is a harsh, often musical systolic aortic murmur, accompanied by a thrill, a decreased or absent second aortic sound, a small pulse, a narrow pulse pressure, and when the electrocardiogram shows left ventricular hypertrophy, and calcification of the aortic valve can be demonstrated roentgenologically. Were one to require all these features, many cases of significant aortic stenosis would be missed. In general, in aortic stenosis any 1 or 2 of these criteria may be absent, but if 3 or more are absent, severe aortic stenosis is unlikely. It must be remembered that the murmur of aortic stenosis may be loudest at the apex or even confined to this area. Its harshness and musical quality often indicate its origin.

It was formerly believed that the direct arterial pressure tracing with prolongation of the systolic upstroke and an anacrotic notch would be a useful adjunct in the diagnosis of hemodynamically significant aortic stenosis. Such tracings, indeed, constitute confirmatory evidence of the presence of aortic stenosis, but not only do not distinguish between mild and severe obstruction at the aortic valve, but may be seen in the absence of any obstruction at the valve. Conversely, severe aortic stenosis may be occasionaly associated with a normal arterial pressure tracing. The presence of aortic stenosis is best documented by demonstrating a systolic pressure gradient across the aortic valve. When obstruction is significant, ventricular pressure exceeds aortic pressure during systole. Since the magnitude of the systolic pressure gradient across the valve is a function of both valve size and valve flow, the degree of aortic stenosis can be determined only if the pressure gradient and cardiac output are measured simultaneously. As with mitral stenosis, catheterization of the left heart is necessary to this end (fig. 5).

Right heart catheterization is of little more than academic interest in the diagnosis of aortic stenosis, but it may have some value in

![Fig. 5. Catheterization of the left heart in aortic stenosis. The pressure tracings obtained from the left ventricle, aorta, and brachial artery of a patient with calcific aortic stenosis have been redrawn and superimposed. The mean systolic pressure in the left ventricle, obtained by graphic integration, was 128 mm. Hg, and the mean systolic pressures in the central aorta and the brachial artery were identical at 68 mm. Hg. Thus the mean systolic aortic pressure gradient was 60 mm. Hg. The cardiac output determined by simultaneous catheterization of the right heart was 5.9 L./min. The calculated systolic aortic valve area was 0.9 cm.² Digital examination at subsequent valvulotomy (Dr. G. W. B. Starkey) revealed severe aortic stenosis.](http://circ.ahajournals.org/.../CLINICAL_PROGRESS)
end of the ductus or extending proximally almost to the pulmonary valve. These occurred almost always in ventricular diastole, less frequently in systole. In patients with ventricular septal defects mixing defects, large or small, oftener in systole than diastole were demonstrated in 7 of 18 cases. With atrial septal defect findings were diagnostic only within sharply margined right atri in 3 of 13 cases. There was 1 demonstrable instance of total pulmonary venous drainage into a left superior vena cava draining into a left innominate vein and another with indentation of the lateral margin of the superior vena cava by a right anomalously draining pulmonary vein.

SCHWEDEL


In rabbits a deep cassette containing 6 films 8 mm. apart is swung manually during injection of radiopaque dye. In order to compensate for the different intensity of the x-rays falling on each film, the more distant films have stronger intensifier screens and are developed longer. The 6 roentgenologic cross-sections of the heart obtained by this method allow recognition of the cardiac cavities, great vessels, and valves with much greater clarity than conventional angiocardiography. Technical improvements will allow the reduction of the swing time to less than 0.1 second and its synchronization with the electrocardiogram, so that systolic and diastolic tomograms can be obtained.

LEPESCHKIN

SURGERY AND CARDIOVASCULAR DISEASE


Recent surgical literature indicates an increasing acceptance of the direct surgical treatment of obstructive arteriosclerosis. The removal of the obstruction and the reestablishment of the vascular channel by resection and replacement by a suitable vascular graft, or by thromboendarterectomy have both been successful. A case of obstructive arteriosclerosis of the right common iliac artery treated by thromboendarterectomy is reported. Maximum benefit was obtained and this benefit has been maintained over a period of 3 years, suggesting that the vessel will remain patent for the duration of the patient’s life and that this area will be the least involved by future arteriosclerotic changes in his total vascular system. Selection of patients with localized or segmental arteriosclerosis and well-developed collateral circulation would seem to be the greatest factor in obtaining a maximum result by thromboendarterectomy.

KITTLE


The authors described their experiences in the surgical closure of a patent ductus arteriosus in a series of 110 operations, performed on patients ranging in age from 7 months to 42 years. In 91 patients the ductus was divided between Potts ductus clamps. In every patient 1 or more antibiotics were given. All patients survived. The authors did not think that division of the ductus was more hazardous than ligation. They pointed out that for the best results in the treatment of this condition, the surgeon, pediatric or medical cardiologist, anesthetist, and radiologist must work together as a team.

ABRAMSON


After attempting various procedures for the correction of transposition of the aorta and pulmonary artery, the author found most satisfactory the technic of utilizing a homologous aortic graft to transpose the inferior vena cava and the right pulmonary veins. This approach was applied successfully in 1 patient.

The operation consisted of dissecting the right pulmonary veins, and the inferior vena cava free and applying a curved coarctation clamp on the latter vessel so that blood flow through it into the right atrium was not occluded. An incision was made in the portion of inferior vena cava held within the coarctation clamp and one end of the aortic graft was anastomosed to the edges of the incision. The open end of the graft was sutured to the lateral wall of the right atrium. Finally, the attachment of the inferior vena cava to the right atrium was divided, causing blood to flow from the inferior vena cava through the aortic graft and into the left atrium. With such a procedure, blood from the inferior vena cava empties into the pulmonary circulation via the left atrium, while blood from the right pulmonary veins reaches the systemic circulation by way of the right atrium.

ABRAMSON


Temporary ligation of the inferior vena cava was
aortic regurgitation is probably significant. It must be kept in mind that peripheral vaso-
dilatation may simulate aortic insufficiency, and vasoconstriction, while actually increasing
regurgitation, may decrease the peripheral signs. Direct arterial pressure tracings give
more accurate information about the absolute level of diastolic and pulse pressures, but do
not permit quantitation of aortic regurgitation. When left heart catheterization demon-
strates only a slight systolic pressure gradient across the aortic valve in the presence of signs
of stenosis and insufficiency, the latter lesion probably predominates and the operation for
stenosis is contraindicated. In the presence of aortic insufficiency, standard aortic valve
formulas overestimate the severity of aortic stenosis, since the increased forward aortic
valve flow (cardiac output plus regurgitant flow) necessitates a larger systolic pressure
gradient across the valve. Thus a method to measure total aortic valve flow is needed before
the relative degrees of aortic stenosis and aortic insufficiency can be quantitatively
assessed by left heart catheterization.

Other Valvular Disease

When mitral stenosis and aortic stenosis co-
exist and both are hemodynamically severe,
the 2 conditions must be corrected at the same
operation. Mitral insufficiency increases opera-
tive risk but does not contraindicate an
operation for aortic stenosis. Such mitral in-
compentence may be improved after ventricular
systolic pressure falls.

Other Factors

Bacterial endocarditis, acute rheumatic
fever, and severe congestive heart failure re-
sistant to treatment represent contraindica-
tions to surgery. Rheumatic activity is less of a
problem in the older age group. Since aortic
stenosis is seen mainly in men of middle age
and older, chronic bronchitis and pulmonary
emphysema coexist not infrequently and should
be evaluated. Functionally significant pul-
monary emphysema increases operative risk
and affects postoperative prognosis adversely.
It may even constitute a contraindication to
operation.

Summary of the Indications for Operation for
Aortic Stenosis

When a patient with clinically pure aortic
stenosis and with no contraindications shows
progressive disabling cardiovascular symptoms,
catheterization of the left heart should be
performed to determine the degree of aortic
stenosis. If stenosis is severe, operation should
be recommended. Contraindications include
coronary atherosclerosis, myocardial infarc-
tion, severe aortic regurgitation, rheumatic
activity, bacterial endocarditis, and intractable
congestive heart failure.

Patients with significant aortic stenosis and
stationary cardiac symptoms may be con-
sidered for operation, provided severe aortic
stenosis can be demonstrated hemody-
namically.

Patients with asymptomatic severe aortic
stenosis may eventually prove to be the best
surgical group, but should not be considered for
operative treatment until more is known about
the long-range effects of aortic valvulotomy.
In all probability the limitations of the calcific
pathologic process itself will continue to make
such “early surgery” improper until valve
replacement is possible.

REFERENCES

1 Ellis, L. B., and Harken, D. E.: The clinical
results in the first five hundred patients with
mitral stenosis undergoing valvuloplasty. Cir-

2 Soulié, P., Di Matêo, J., and Azérâd, J.: Rés-
sultats de la commissurotomie pour retrécis-
mement mitral (à propos de 213 interventions).
Bull. et mém. d. hôp. de Paris nos. 21–22, 742,
1955.

3 Bailey, C. P., and Bolton, H. E.: Criteria for
and results of surgery for mitral stenosis. Part
II: Results of mitral commissurotomy. New

4 Janton, D. H., Davila, J. C., and Glover, R. P.:
Status of fifty patients four and a half to seven
years after mitral commissurotomy. Cir-
culation 14: 175, 1956.

5 Grant, R. T.: After histories for ten years of one
thousand men suffering from heart disease.
Heart 16: 275, 1933.

6 Wilson, J. K., and Greenwood, W. F.: The
natural history of mitral stenosis. Canad.
I hold every man a debtor to his profession; from the which as men of course do seek to receive countenance and profit, so ought they of duty to endeavor themselves by way of amends to be a help and ornament thereunto.—Francis Bacon, 1561–1626.
Selection of Patients for Mitral and Aortic Valvuloplasty
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