Editorial
The Surgical Treatment of Acquired Valvular Disease

The surgical correction of mitral stenosis can be excellent, of aortic stenosis good, of tricuspid stenosis simple. Any or all of these can be corrected at one operation if necessary. The surgical correction of organic insufficiency of these valves is now only palliative. Functional mitral, tricuspid, and pulmonic insufficiency respond to correction of the primary condition. Functional tricuspid stenosis like insufficiency, responds to correction of the associated mitral stenosis.

Pure lesions and all combinations must be considered individually. In each instance, certain relevant pathologic, clinical, and hemodynamic features must be mentioned to clarify the selection of patients.

MITRAL STENOSIS

Experience with mitral stenosis has clarified its diagnosis, life cycle, surgical risk, and rehabilitation. To define risk, patients suffering from mitral stenosis have been divided into 4 groups broadly defined as follows: Group I: patients with the murmur of mitral stenosis but no disability; auscultatory disease of no hemodynamic significance. Group II: individuals with mild nonprogressive limitation. Group III: those with pure mitral stenosis and progressive symptoms. Compensation is disintegrating. Group IV: patients who are in the terminal or late phases of the life cycle, generally with refractory congestive failure. Often they have irreversible damage to the lung, liver, or myocardium. Ellis has carefully studied the first thousand valvuloplasties performed by Harken's group.

The surgical mortality has fallen dramatically. Group III patients had a 0.6 per cent mortality in the second 500 operations. The mortality remained high (20 per cent) for the same period among the group IV patients. Not only has the mortality in group III patients reached an enviable level comparable to other general surgical procedures but rehabilitation is better before there is damage to the lungs, liver, and myocardium. Useless delays are often counseled because of possible rheumatic activity, aortic insufficiency, subacute bacterial endocarditis, and thyrotoxicosis. These diseases are readily established or eliminated beyond reasonable doubt while the patient is being prepared for surgery.

A final echelon of confusion has arisen due to the occurrence of mitral stenosis without a murmur. Happily, this has led to more careful auscultation in better positions and with a variety of stethoscopes so that this event has become less common. Most careful auscultation has clarified ancillary physical evidence of mitral stenosis and conversely has shown that the loudest diastolic murmurs may occur in dominant mitral insufficiency. This loud diastolic murmur of insufficiency is due to the increased amount of blood flowing over the valve during diastole.

Although 530 of the first thousand surgical patients studied by Ellis had atrial fibrillation, this arrhythmia is still mentioned as a surgical contraindication. Incidentally, preoperative reversion to normal sinus rhythm is ill advised, for they often fibrillate again.
after surgery. The total risk of embolus is probably less in proceeding directly to operation. It is better to flush out thrombi at operation rather than to risk embolus during medical reversion. After surgery it may be possible to restore normal sinus rhythm permanently.

Valvuloplasty significantly reduces the danger of embolism in mitral stenosis, presumably because stasis is reduced and the appendage is removed. Because repetition of embolization is common, one episode may be a substantial factor in favor of immediate operation. Repeated emboli within a period of a few weeks may constitute an urgent indication for valvuloplasty, and repeated embolization within a space of a few days in spite of anticoagulant therapy defines a surgical emergency unless the patient's general condition precludes surgery. Even in such surgical emergencies embolization has always been stopped by valvuloplasty and coincident appendicectomy.

The danger of embolization at surgery has been reduced by 3 factors. First, flushing the atrium evacuates thrombi from the appendage. Second, occlusion of the cervical vessels for appropriate intervals after fracturing through calcifie areas in the valve reduces the chance of cerebral emboli by shunting blood past the brain to silent areas. Third, experience helps the surgeon decide where to fracture in order to avoid mobilizing calcifie fragments.

Just as experience has led to better fracture with fewer emboli, it has improved mobilization of leaflets without the production of regurgitation. Indeed, regurgitation may be reduced by valvuloplasty. The propriety of posterior commissural opening is a technical consideration not within the scope of this discussion. There is a place, however, for such fracture or incision. At times this posterior fracture must be avoided to avoid regurgitation; at times it must be used in order to effect satisfactory valvuloplasty. Failure to obtain good valvuloplasty initially is the most common cause for poor surgical results.

Re-operation must be considered when any patient who has had pure mitral stenosis fails to demonstrate or maintain improvement.

The safe conduct of a patient with severe mitral stenosis through pregnancy is a real medical challenge. A good medical regimen and bed rest will allow most patients with auscultatory disease and many with hemodynamically significant disease to negotiate pregnancy safely. However, there are patients who develop pulmonary edema or congestion and progressive symptoms in the first trimester where pregnancy is unsafe for mother or baby. A persuasive statistical case can be marshaled for the interruption of pregnancy and the correction of mitral stenosis later. This does not take several factors into consideration. First, abortion with subsequent valvuloplasty necessitates 2 operations. Second, experience indicates that valvuloplasty can be carried out safely if the patient has tight mitral stenosis and the pregnancy is in the first trimester, (a greater risk may exist, yet be justified later). Third, religious factors may influence the decision. Fourth, many mothers simply want their babies. How one weighs this last factor must be an individual matter.

Three warnings should be sounded in connection with mitral stenosis and pregnancy. The diagnosis of pure mitral stenosis during pregnancy is notoriously unreliable. The increased blood flow of pregnancy over an only slightly roughened valve may suggest stenosis when the disability is actually due to something else. A reliable diagnosis of stenosis before pregnancy is therefore virtually essential for a safe decision. The danger of producing congenital anomalies suggests that the end of the first trimester is best for the correction of stenosis. The pregnancy is also more secure. Finally, delivery by cesarean section is inadvisable after successful valve surgery unless specifically indicated by such factors as cephalopelvic disproportion.

Patients past 50 have been considered too old for valvuloplasty by some clinicians. We

*Specifically, these women would be classified as group III before pregnancy.
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have operated on approximately 200 patients between the ages of 50 and 70. These patients have enjoyed results comparable to younger patients. The incidence of heavily calcified valves is higher; the occurrence of other complicating diseases is more prevalent. Thus, if a patient has mitral stenosis, its correction must be considered on its individual merits in that patient.

Aortic Stenosis

There are definite indications for surgical intervention for aortic stenosis. Earlier experience with the valvulotome through the left ventricle was disappointing. The results were not comparable to the transaortic leaflet mobilization. In this operation, a small Ivalon (polyvinyl alcohol) operating tunnel is sutured to the side of the base of the aorta to allow direct digital manipulation. A valvulotome may also be used if necessary. I am convinced that this operation has advantages over available direct-vision procedures. This operation can be careful, deliberate, and meticulous without an open technic per se with hypothermia or pump-oxygenator. It also affords an opportunity to assess valve mobility under conditions of flow. Formidable as many calcific valves look at autopsy, experience indicates that some mobilization is usually possible.

Comparison of surgical patients with patients not having surgery clarifies the place for operation. Among our first 80 modern transaortic operations there have been 13 surgical deaths. Seven operative deaths occurred in the first 20 operations; among the second 20 operations there were 4 deaths; in the last 40 operations there have been but 2 operative losses. Apparently, this operative morality may now be as low as 5 per cent. Eight late deaths indicate that some patients do not do well. However, more than half return to work. The salvage in this group is striking when compared with a personally observed “control” group of 52 patients who, for one reason or another, failed to have surgery. Of these 52 patients who did not have surgery 47 were dead within 6 months after surgery had been advised.

This formidable experience is a result of the selection of patients largely on the basis of left ventricular failure and also because it includes some patients who succumbed before surgery could be done. Nevertheless, it indicates what a devastating disease aortic stenosis can be when it is hemodynamically significant to the point of left ventricular failure. Left ventricular failure as manifested by progressive dyspnea in spite of adequate medical therapy seems to be an urgent indication for intervention. Persistent angina and troublesome syncope may also justify surgery, although Ellis and his colleagues have shown that some of these patients may survive for years. Contrariwise, left ventricular failure or atrial fibrillation is very ominous. Ellis further found that the onset of pain or syncope during atrial fibrillation or failure was generally followed by death within days or weeks. This better understanding of the life cycle of aortic stenosis interpreted in the light of the results of surgery clearly defines a place for operative intervention. The left heart catheterizations and hemodynamic studies of valve gradients, cardiac outputs, and dye dilutions carried out on many of these patients by Dexter and his group, indicate that this surgery does not compare favorably with valvuloplasty for mitral stenosis. However, even in cases where left heart catheterization is unimpressive, clinical palliation may be excellent.

Aortic and mitral stenosis can be corrected at the same operation if necessary. The severity of aortic and mitral stenosis in combination can best be evaluated by left heart catheterization. Rarely should surgery be performed on patients with multivalvular problems without such studies. This includes not only valve gradients but cardiac output and dye-dilution studies. Formerly, pressures and gradients were measured at the operating table but preoperative left heart catheterization has largely replaced such studies.

Tricuspid Stenosis

The surgical correction of tricuspid stenosis is technically easy but rarely indicated.
Aortic and Mitral Regurgitation

The surgical correction of aortic and mitral regurgitation has not been so successful as that of stenosis. In aortic insufficiency the Hufnagel valve presumably reduces ventricular work, but at the same time, it reduces diastolic perfusion of the coronary arteries. We abandoned this operation some years ago in favor of a direct circumclusion procedure, which reduces the size of the base of the aorta below the coronary ostia. This operation involves careful dissection of the base of the aorta; isolation of the coronary arteries and anchoring a tapered band about the aorta in a zone of leaflet insertion below the coronary ostia. Extensive calcification makes this operation impossible.

Just as the pathologic process in aortic stenosis may limit the quality of surgical repair, some patterns of aortic insufficiency and combinations of stenosis and insufficiency are clearly not amenable to surgical correction where only the existing damaged parts are used. Prosthetic valves must be developed. The objection to the Hufnagel valve is that it reduces diastolic pressure and coronary perfusion because of its position in the descending aortic arch. The obvious answer is to place the valve at the normal valve site. There are serious technical problems in designing such a valve and in placing it properly so as to avoid coronary obstruction and cerebral embolus. This need and these difficulties have again made us look hopefully at a prosthetic aorta containing a valve, originating from the apex of the left ventricle and inserting into the lower thoracic aorta, as suggested by Donovan and Sarnoff. Aortic stenosis constitutes the first place for such a by-pass. With ventricular pressures reduced to normal levels the stenotic orifice would probably cease to have significant function. In free aortic insufficiency or mixed stenosis and insufficiency, a similar by-pass could be used if the natural outflow tract or aortic base were closed. This should not be an insurmountable technical problem.

The surgical correction of mitral incompetence has been a troublesome and frustrating pursuit. A review of certain basic hemodynamic and morphologic concepts makes the clinical picture and the surgical challenge clearer. It also forms a basis for reviewing past and present operations. The morphologic lesion in mitral insufficiency may be an absolute or relative loss of valve leaflet substance, or both. The valve may fail to close because of small leaflets or an enlarged annulus. When small leaflets allow regurgitation, it is compensated by a greater volume of ejected blood, and thus greater ventricular chamber size, which in turn may cause an increase in the size of the annulus and greater relative insufficiency. This cycle may also be initiated by primary myocardial disease.

Two other mechanisms in this self-aggravating cycle point the direction for definitive surgery and present palliation. First, there is atrial herniation of the valve ring that causes the most marked incompetence posteriorly. Second, herniation renders the chordae tendineae relatively short and interferes with leaflet closure. The normal position of the mitral annulus is just within the upper portion of the left ventricle. Closure of this valve involves the reduction of annulus size by ventricular systole, the relaxation of the leaflet edges and upward movement of the ventricle and papillary muscles to allow leaflet closure and torsion of the chordae tendineae to secure closure. The leaflets are in direct continuity not only with the annulus but with the atrial endocardium. Any systolic jet impinges most forcibly on the adjacent atrial wall, i.e., the atrial insertion at the posterior atrioventricular groove. This force dilates the atrium posteriorly and displaces the annulus up and over the ventricular rim, producing the direct herniation of the valve complex. As the annulus slides over the ventricular rim, the leaflets are shortened and regurgitation is increased locally, demanding further ventricular dilatation to compensate for increased reflux. Thus a vicious cycle is accelerated. Finally, the upward migration of the valve puts traction on the chordae in one direction and the dilatation of the ventricle pulls them in the
opposite direction. Thus taut, the chordae cannot relax to allow leaflet closure in systole and reflux is again aggravated. All these factors result in an excessive increase in ventricular fiber length, loss of myocardial efficiency, and ventricular failure.

Appreciation of these factors in the production and aggravation of insufficiency indicate the folly of suturing leaflets together to correct regurgitation. If the existing valve components are to be utilized in reconstructing a competent valve, the leaflets must be mobile. The annulus must be replaced to its normal position within the ventricle so that the ventricular action supports closure and the chordae tendineae can relax in systole. As the hernia is reduced and the leaflets are replaced within the ventricle, the posterior shortening of leaflets is corrected.

It is obvious how bed rest may reverse all of these aggravating factors. The reduced stroke volume can reduce chamber size, thus reduce annulus diameter and increase leaflet competence and so on. However, at some point the aggravation perpetuates itself in a malignant form unless it is stopped by some surgical maneuver. This operation must either replace the defective valve or, utilizing existing valve components, correct the deficiencies, i.e., leaflet size, annulus size and position, chordae length, and secondarily, ventricular myocardial fiber length. Formerly attempts were made to substitute substance for relative or absolute leaflet loss by baffles. This was hemodynamically attractive but the incidence of embolus precluded their use in patients early enough for maximal efficiency. In the course of these and other operations, it was noted that mild to moderate degrees of regurgitation could be corrected by digital distortion of the mitral annulus by posterial pressure with the finger on the outside of the heart in the atrioventricular groove. This maneuver was even more effective if the atrial wall was invaginated slightly toward the ventricular chamber, thus reducing the valve herniation. This lengthened the leaflets, relaxed the chordae tendineae, and if the annulus was compressed into an ellipse simultaneously, the shortened leaflets could close the orifice still more effectively. This suggested a mechanism of improving mild to moderate regurgitation. The problem was to get a substance to maintain the same force as the finger. Various substances including luteite, Ivalon (polyvinyl alcohol), and steel wire were used. None of these materials seem to maintain their position as well as Gel-Foam® compressed to a hard, nonabsorbable state. As this extrinsic baffle is placed, the correction of regurgitation is noted by a finger in the heart. Experience with more than 200 of these extrinsic baffles indicates that the procedure is simple, safe, and useful in correcting those mild to moderate degrees of incompetence often encountered at valvuloplasty for stenosis.

When the dominant lesion is marked regurgitation, the available surgical maneuvers leave much to be desired. Open correction is encouraging but is still in the experimental stage. D’Avila and Glover have developed a circumclusion operation that is attractive in several aspects. It cannot reduce the herniated complex, thus the disadvantage of relative shortening of the chordae tendineae remains. There are additional technical and clinical reasons that temper enthusiasm for this procedure. The markedly disabled patient who should be benefited if a procedure is sound, does not tolerate this circumclusion. We abandoned this procedure 2 years ago. D’Avila and Glover have now tried this circumclusion on a more favorable risk group, and report salutary effect. If their reasons for moving to good risk patients were valid, operations on patients with terminal aortic valve disease should fail; such has not been our experience. One can only conclude that definitive surgery for severe degrees of mitral insufficiency has not yet been established.

In conclusion, this wide spectrum of operations complicates the selection of patients. The old platitude "the patient is doing very well" becomes only a relative comment. If
pure mitral stenosis is causing no limitation and is not damaging the lungs or myocardium by pulmonary hypertension, "doing well" is acceptable. We are not ready for prophylactic valvuloplasty as in closure of the patent ductus. Valvuloplasty is not quite curative; indeed, when poorly done, stenosis may recur. However, as soon as the patient with mitral stenosis "begins to have to give up things in life," he is no longer "doing well" and surgery must be considered. When symptoms are progressive, surgery must be urged. The place for surgery in pregnancy, after embolus, and in the terminal phases of the life cycle of disease has been clarified. "Following a patient," from group III with an operation risk of 0.6 per cent into group IV, with a 20 per cent mortality, is condemned.

These new medical responsibilities have sharpened all diagnostic technics. The history must discover concealed but serious limitations such as those in sex activity. Progressive dyspnea must be recognized. Dyspnea that does not seem to be progressive may appear static due to self-imposed or physician-imposed restriction. If the patient admits of no limitation, one must detect the limitation unrecognized because of insidious onset. On the other hand, one must not be confused by limitation imposed by a physician, or so-called iatrogenic disability. In cases where the disability is difficult to assess, right or left heart catheterization may help measure the hemodynamic disturbance.

Aortic stenosis may cause angina, syncope, atrial fibrillation, and all degrees of left ventricular failure. Surgery has a place when the symptoms are progressive, when the heart size increases, or when the left heart catheterization shows a critical valve size or an intolerable gradient. Surgery is urgently indicated when there is left ventricular failure or progressive dyspnea in spite of a good medical regimen. Minor degrees of aortic insufficiency associated with stenosis may improve with good aortic valvuloplasty.

In aortic insufficiency, the surgical treatment is incomplete and only palliative. This makes recognition of the role of surgery all the more difficult. There are minor hemodynamic problems that certainly are much better handled by waiting for better operative procedures. Rigid medical treatment may be life-saving. The effective "medical regimen" involves measured salt restriction, proper use of digitalis, diuretics, drug prophylaxis, and work tolerance assay and regulation. Overt congestive failure is late and ominous. A diuretic response, before congestive failure becomes obvious on physical examination, is a critical method of detecting occult failure. In the presence of overt or latent failure, surgery must be considered.

Severe mitral regurgitation will be better treated by a more definitive operation in the future but minor degrees of regurgitation must not prevent a patient from receiving valvuloplasty for stenosis. In the course of the surgery, available therapy or "strategic retreat" with the extrinsic baffle is useful. The simplest operation for severe pure mitral regurgitation appears to be D'Avila's and Glover's mitral circumclusion; the most definitive: open operation.

The surgical correction of acquired valvular disease is routinely followed by meticulous medical treatment for some months. This course is important during the period of myocardial adjustment to new hemodynamic factors. This need does not mean that surgery has failed any more than the persistence of a murmur. The physician will do well to reduce the medical program gradually.

Surgery has complicated but stimulated the whole field of cardiology.

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