This paper discusses the indications for surgery in mitral valve disease by comparing the natural history of the disease, as it can best be determined from the literature, with the results of various surgical procedures. It may be stated at the outset, however, that there are no controlled studies comparing medical and surgical treatment nor are they likely to be carried out in the near future.

**Mitral Stenosis**

1. **Detection.** While the auscultatory findings of mitral stenosis are characteristic, many patients go undetected by routine clinical examination; it is, therefore, important to be aware of the special syndromes with which they may present (Table 1).

Mitral stenosis is much more common in women and should be looked for particularly in those who are young or middle-aged. The condition is predominantly a sequel to rheumatic heart disease which is prevalent among large families from lower socioeconomic groups. The availability of M-mode echocardiography in recent years has been of great help in the detection of mitral stenosis, since this procedure has nearly 100% sensitivity and the specificity is almost as good. With the two-dimensional echocardiogram the degree of stenosis can be measured with great precision by mapping out the valve area. Echocardiography has thus eliminated the need for cardiac catheterization either for the detection or quantification of mitral stenosis, and cardiac catheterization is now employed only preoperatively for the detection and assessment of associated lesions.

2. **Natural history.** Bland and Jones in their classical 20-year study of 1,000 patients with acute rheumatic fever showed the extreme variability in the rate of progression of mitral stenosis in different patients. Some patients developed mitral stenosis within 5 years after the first attack of rheumatic fever while others were relatively well even 50 years later; however, a composite average can be deduced from this and other studies. In the preantibiotic era, mitral stenosis was clinically detectable approximately 10 years after the onset of acute rheumatic fever, symptoms were minimal in the next 10 years unless pregnancy or complications occurred (for example, endocarditis or atrial fibrillation), and in the next 20 years symptoms increased progressively with development of pulmonary hypertension, heart failure, and systemic embolism, leading to death, approximately 40 years after the onset of rheumatic fever, at an average age of 48 (Fig 1).

It is my impression that mitral stenosis today shows a slower rate of progression in the industrialized countries, mainly because the use of antibiotics has considerably improved the rate of recurrence of rheumatic fever and the treatment of endocarditis.

One of the most useful studies of the natural history of mitral stenosis is by Olesen and Baden who classified their patients according to the New York Heart Association (NYHA) classification and compared the non-surgical groups seen during the years 1933-1950 with the initial 165 consecutive patients operated on during the years 1950-1955 (Fig 2). Most studies of surgical treatment of mitral stenosis have
TABLE 1
Some Presenting Syndromes in Mitral Stenosis

- EXERTIONAL DYSPNEA OR PULMONARY EDEMA WITHOUT GENERALIZED CARDIOMEGALY
- ATRIAL FIBRILLATION
- SYSTEMIC EMBOLISM (STROKE, MYOCARDIAL INFARCTION, HEMATURIA, ETC.)
- HEMOPTYSIS WITHOUT PULMONARY INFECTION
- RESTRICTIVE AND OBSTRUCTIVE LUNG DISEASE WITHOUT SMOKING HISTORY

used Olesen and Baden’s non-surgical patients for comparison. In NYHA Class II patients with normal sinus rhythm the ten-year survival was 80%. At the other extreme over 50% of Class IV patients were dead within five years and all were dead within eight years.

3. Choice of operations and results. The possibility of correcting the mechanical problem of mitral stenosis surgically was first discussed around the turn of the 20th century. The first attempts at surgical correction were made in the 1920s, but mitral valvotomy has come to be recognized as a generally acceptable method of treatment only since 1948. Today several different surgical procedures are available for the repair of mitral valve disease, some of the more common being listed in Table 2.

The operative and postoperative morbidity and mortality rates are very low for mitral commissurotomy but considerable when mitral valve replacement with a prosthesis is necessary. It is important, therefore, to determine preoperatively whether the patient with mitral stenosis will require commissurotomy or valve replacement. Table 3 gives some of the signs that determine this decision. Successful mitral com-

Asymptomatic

Mild

Moderate

Severe

Total Disability

Death

Zone of Bacterial Endocarditis

Pregnancy

Increasing Dyspnea

Pulmonary Hypertension

Cardiac Failure

Zone of Embolism

YEARS AFTER ACUTE ATTACK

Normal

Mild

Moderate

Severe

4-6 cm²

1.5 - 2.5 cm²

1.1 - 1.5 cm²

0.6 - 1.0 cm²

Rest

LA N

PA N

CO N

Exer

LA ↑

PA ↑

CO ↑

LA ↑

PA ↑

CO ↑

LA ↑

PA ↑ or ↓

CO ↑ or ↓

Fig 1—Natural progression of mitral stenosis. Clinical course, pathology and hemodynamic changes are correlated. Valve area is indicated below each diagram of valve. CO = cardiac output; Exer = exercise; LA = left atrial pressure; N = normal; PA = pulmonary arterial pressure; ↓ = decreased; ↑ = increased.

48 / HASSAN: SURGERY FOR MITRAL VALVE DISEASE
missurotomy is not possible if significant regurgitation accompanies stenosis or if the valve is heavily scarred, markedly deformed, or calcified and immobile. Attempts at valvotomy in calcified valves result in fracture of the valve with resultant insufficiency rather than cleavage of the commissures. Lack of valve mobility is evident clinically by the absence of an opening snap and the presence of a muffled first heart sound; calcification can be detected by fluoroscopy and echocardiography. Repeated commissurotomies also cause extensive scarring and deformity which necessitate valve replacement.

Olesen and Baden showed that 'closed'

**TABLE 2**

Common Operations Available for Mitral Valve Disease

- COMMISSUROTOMY
  - CLOSED
  - OPEN
- VALVE REPAIR
  - ANNULOPLASTY
  - VALVULOPLASTY
  - CARPENTIER RING
- VALVE REPLACEMENT
  - CAGE AND BALL VALVE
  - TILTING DISC VALVE
  - PORCINE TISSUE VALVE
TABLE 3
Signs Indicating Probable Need for Valve Replacement in Mitral Stenosis

- INTENSITY OF S1 OR OPENING SNAP REDUCED
- SIGNIFICANT MITRAL INSUFFICIENCY
- DENSE CALCIFICATION OF MITRAL VALVE
- PRIOR MITRAL SURGERY

mitral commissurotomy (that is, commissurotomy without cardiopulmonary bypass or 'open'-heart surgery) improved survival in all but minimally symptomatic patients in normal sinus rhythm. Long-term survival was adversely affected by the presence of preoperative pulmonary hypertension or postoperative mitral insufficiency. Ellis, Harken and Black reported essentially similar results in a much larger series of 1,000 surgical cases.

In the United States, most institutions use open mitral commissurotomy (that is, with cardiopulmonary bypass) as the procedure of choice for mitral stenosis. The results of open commissurotomy are probably superior to the closed operation, although it is a more expensive procedure. Improvements in surgical technique have markedly reduced the incidence of systemic embolization and mitral insufficiency, which were the principal dangers of mitral commissurotomy in earlier days. The mortality from mitral commissurotomy today is less than 1% when the operation is done at an optimal time and most survivors experience improvement in their symptoms.

When mitral stenosis can be treated with commissurotomy, the operation should be recommended as soon as symptoms interfere with the normal enjoyment of life or at the earliest signs of complications. In a substantial minority of patients, the valve restenoses after several years with recurrence of symptoms. Additional surgery should be considered in these patients.

The other types of mitral valve surgery will be discussed with the treatment of mitral insufficiency.

Mitral Regurgitation

The causes of mitral regurgitation are numerous; however, it is usually caused by congenital heart defects in young children, rheumatic heart disease or mitral valve prolapse in young adults, and coronary artery disease in older patients. The systolic apical murmur radiating into the axilla is usually easy to detect and is almost diagnostic of mitral regurgitation, although occasional patients with aortic stenosis have their murmur referred to the mitral area. Rupture of chordae tendineae to the posterior mitral leaflet may produce a murmur loudest at the sternum, simulating aortic stenosis. After mitral valve replacement, significant mitral regurgitation may occur silently but should be suspected if left ventricular failure or hemolytic anemia develops unexpectedly.

Detailed studies of the natural history of mitral regurgitation according to severity of the lesion or staging by symptoms are not available. Some impressions can be drawn from the prospective study of rheumatic fever by Bland and Jones. The prognosis of asymptomatic, mild, chronic mitral regurgitation without cardiomegaly is no different from that of the normal population; however, when significant symptoms are present with moderate-to-marked cardiomegaly, life expectancy is considerably shortened. In Rapaport's experience approximately 20% of the patients with mitral regurgitation were dead within five years and 40% within ten years after their initial diagnosis.

Another consideration in mitral regurgitation regarding prognosis is the cause of the lesion. Most patients with mitral valve prolapse (click-murmur, Barlow syndrome) have a nonprogressive lesion. Mitral regurgitation secondary to chronic left ventricular failure and dilatation (for example, cardiomyopathy, hypertension) usually does not respond well to surgical treatment. In contrast with chronic mitral insufficiency, acute mitral insufficiency is very poorly tolerated and leads to rapid heart failure. Acute mitral regurgitation usually results from rupture of papillary muscles, chordae tendineae or valve leaflets due to infarction, infection or trauma, and occasionally spontaneously. In acute cases more attention should be paid to the severity of the hemodynamic lesion, as determined by physical signs and cardiac catheterization, than to the symptoms.

The combined lesion of mitral stenosis and regurgitation is usually due to rheumatic heart disease and is treated like mitral regurgitation because most patients require mitral valve replacement.

Mitral Valve Repair

The concept of repairing the valve instead of replacing it with a prosthesis is extremely at-
tractive, particularly for younger patients. It avoids the problems of thromboembolism, infection and hemolysis consequent upon the introduction of a foreign body into the bloodstream. The proponents of valve repair claim that it is feasible in a majority of patients with mitral regurgitation, but others have largely avoided it. Several different techniques have been described. Annuloplasty reduces the size of the valve orifice by plication of the annulus fusing the peripheral portions of the mitral commissures. Complete obliteration of mitral insufficiency is frequently not possible by this technique and postoperative dilatation of the mitral annulus may necessitate valve replacement at a later date. Valvuloplasty is a spectacularly successful and curative operation for many cases of ruptured chordae tendineae. The redundant untethered portion of the posterior leaflet is obliterated by pleating with sutures. The Carpentier ring is a slightly irregular bicuspid stainless steel ring wrapped in a teflon collar for sewing which has been recently introduced as an aid to valvuloplasty. The ring prevents further dilation of the valve, and the sewing collar is used to anchor the sutures and remodel the deformed valves to approximate normal anatomy. Risk of thromboembolism and endocarditis is not significantly increased with the use of the Carpentier ring. At present, valve repair should be strongly considered in younger patients and in those with mitral insufficiency secondary to rupture of the posterior leaflet chordae tendineae. With continuing improvements in technique, valve repair is likely to become possible in a larger proportion of patients. The risk of valve repair is minimal, and marked symptomatic improvement occurs after successful surgery, but it is not possible to decide preoperatively whether repair or valve replacement will be necessary in a specific patient. The indications for valve repair are therefore the same as for valve replacement as discussed below.

**Mitral Valve Prostheses**

Three main types of prostheses are currently in use (Fig 3). Various models of the Starr-Edward heart valves are examples of the cage-ball type of prosthesis. Blood flows turbulently around the occluding ball in contrast to the laminated flow in the normal valve. Long experience

---

Fig 3—Three commonly used mitral valve prostheses. A. Starr-Edward Model 6120; B. Bjork-Shiley; C. SGP Hancock Porcine Model 342.
with these valves has shown them to be durable and reliable. The main problem is thromboembolism which is reduced but not eliminated by anticoagulants. Traumatic hemolysis may occur in models of the valve with cloth-covered struts. Another problem is the very loud closing click with some models. This sound can occasionally be heard by the unaided ear across the room from the patient. The incidence of infection of the prosthesis is probably similar to the other prosthetic valves.

Tilting disc valves, such as the Björk-Shiley, have a more physiological blood flow and a low profile so that the valve does not occlude the left ventricular cavity. The major advantage of this valve is that the sewing ring is not disproportionately larger than the internal lumen and these valves are therefore associated with the lowest transvalvar gradients among all prosthetic heart valves. However, anticoagulation is needed and the closing click can be quite noisy. This is the prosthetic valve of choice in patients with a small mitral valve annulus.

Glutaraldehyde-treated porcine heterografts are becoming increasingly popular because of low thrombogenicity even without anticoagulation. These valves consist of porcine aortic valve tissue mounted on a man-made frame. Flow is central, very much as in the normal valve, but the sewing ring is considerably larger than the internal lumen, and large transvalvar gradients may be present postoperatively, particularly with the smaller-sized sewing rings. Long-term durability is another major concern. Nevertheless, this is the prosthesis of choice when anticoagulation is contraindicated.

Characteristics of the three valves discussed above are compared in Table 4.

**Results of valve replacement.** Comparative studies of different types of valve prostheses are not available at present. It appears, however, that the operative mortality for the different types of prostheses mentioned above is roughly comparable, and the risk factors for operative mortality and long-term results are also similar. The most important risk factors for early operative mortality are advanced heart failure and marked cardiomegaly, particularly left atrial enlargement (Fig 4). The long-term results are adversely affected in patients with marked left atrial enlargement or pulmonary hypertension. Most survivors of the operation show symptomatic improvement, but their survival curve does not parallel that of the general population (Fig 5). Young patients with severe mitral regurgitation show a remarkable reduction in cardiac size following successful surgery, whether valve repair or valve replacement. Mitral valve replacement is recommended in all patients with significant symptoms, that is, NYHA Class III or IV. It is also indicated in acute severe mitral regurgitation and significant mitral regurgitation with progressive cardiomegaly or pulmonary hypertension, even when symptoms are less impressive (Table 5). It is advisable not to wait until advanced stages of heart failure, cardiac enlargement, or pulmonary vascular changes occur, but unfortunately patients are all too frequently in this condition when they first seek, or are referred for, valve surgery.

In summary, the natural history of mitral valve disease is discussed and compared with various surgical procedures. Life expectancy is considerably shortened when mitral stenosis is accompanied by significant symptoms or complications. Mitral commissurotomy is a relatively benign procedure (< 1% mortality) which improves survival and symptoms in these patients. Chronic mild mitral regurgitation is tolerated well, but progressive cardiac dysfunction occurs when regurgitation is severe, particularly if it develops acutely. Valve repair can be performed in some cases with a low mortality and very low postoperative morbidity. Most patients with incompetent valves require valve replacement in which the average surgical mortality is approxi-
Fig 4—Early operative mortality in isolated mitral valve replacement with Model 6120 Starr-Edward prosthesis, Mayo Clinic 3/66 to 1/72; A = mortality according to preoperative NYHA classification; B = mortality according to preoperative left atrial (LA) size.

Fig 5—Late survival and systemic thromboembolism in surgical survivors of isolated Starr-Edward mitral valve replacement with model 6120 prosthesis compared to normal life expectancy.
TABLE 5
Indications for Valve Replacement in Mitral Regurgitation

- NYHA CLASS III OR IV
- SIGNIFICANT ENLARGEMENT OF LEFT ATRIUM OR VENTRICLE
- PULMONARY HYPERTENSION
- ACUTE SEVERE MITRAL REGURGITATION

mately 5%; thromboembolism is the most seri­ous postoperative problem.

Surgical mortality is markedly increased in advanced heart disease (NYHA Class IV) and surgical treatment should therefore be consid­ered in all patients with mitral valve disease be­fore this stage is reached.

Figure 1 is taken in part from Proceedings of the Royal Society of Medicine (60:1009–1015, 1967) and the American Journal of Cardiology (35:221–227, 1975) by permission.

Figure 2 is adapted from the Scandinavian Journal of Thoracic and Cardiovascular Surgery (3:119–124, 1969) by permission.

Figure 4 adapts data found in Barnhorst et al, American Journal of Cardiology (35:228–233, 1975).

Figure 5 is adapted from the American Journal of Cardiology (35:228–233, 1975) by permission.

REFERENCES


