

Mitral valve repair

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Patients with chronic mitral regurgitation present with a wide spectrum of symptoms but generally fit one of two categories. There are those with a murmur and relatively asymptomatic (functional class I or II), or those in congestive cardiac failure from long-standing regurgitation, who are usually quite ill (functional class III or IV). The diverse aetiologies include coronary disease, rheumatic disease, infection, trauma, congenital anomalies, myxomatous degeneration and mitral valve prolapse. Most adult causes are classified as ischaemic, rheumatic or degenerative.

Investigation

History and physical examination substantiate the aetiology, and the physical examination documents enlargement of the heart and a harsh holosystolic apical murmur. ECG and CXR establish cardiomegaly. Serial CXR examinations may establish progressive enlargement of the heart over time (especially in asymptomatic patients).

Transthoracic and transoesophageal echocardiography are major diagnostic aids. As well as documenting progressive left ventricular dysfunction using systolic and diastolic volume indices, transoesophageal echo (TOE) can determine precisely whether valve repair is feasible and exactly which techniques of repair will be required.

In patients who are asymptomatic or, at worst, functional class II, the decision for surgical therapy depends on documentation of enlarging heart size and progressive left ventricular dysfunction. If a patient has a murmur but normal heart size on noninvasive testing, no further therapy is indicated. The patient should be followed at six monthly intervals with two-dimensional echocardiography.

On the other hand, patients with an enlarged heart and left ventricular dysfunction should proceed to cardiac catheterisation and coronary angiography if they are over 40 years old or have chest pain. Symptomatic patients with congestive cardiac failure or NYHA III or IV who have documented MR on echocardiography should progress immediately to cardiac catheterisation and coronary angiography.

Indications for surgery

Operation for symptomatic patients with chronic mitral regurgitation (NYHA class II or greater) should be considered before irreversible left ventricular systolic dysfunction

develops. The degree of deterioration in left ventricular contractility determines for the most part the patient's prognosis and the optimal time for operation. If the left ventricular ejection fraction is $>60\%$, 10 year survival after surgery is 72%. If the left ventricular ejection fraction is $<50\%$, 10 year survival after surgery is 32%.

Clinically, loss of contractile function is extremely difficult to measure with any precision as mitral regurgitation enhances left ventricular emptying by reducing the impedance to ventricular emptying. Almost half the regurgitant volume is ejected into the left atrium before the aortic valve opens. Hence usual parameters such as ejection fraction and end diastolic dimension become difficult to interpret. The left ventricular volume overload and the low impedance during systole may give a large ejection fraction (usual normal 58-74%) and less than 60% may indicate dysfunction.

Likewise, other parameters of left ventricular function such as end diastolic dimensions and fractional circumferential fibre shortening can be normal even with severely depressed left ventricular systolic function. Left ventricular end systolic diameter as measured at echocardiography is a widely employed surrogate that reflects changes in systolic function. It is independent of preload and varies directly and linearly with afterload.

Therefore, the larger this dimension the worse is the systolic function and it has proven to be very useful in mitral regurgitation as a predictor of left ventricular function. Values greater than 5.0cms are associated with poor operative outcome and values less than 4.0cms have excellent postoperative outcome.

New York Heart Association (NYHA) Functional Classification

- Class I: Ordinary activity does not cause undue fatigue, palpitation, dyspnoea or anginal pain.
- Class II: Ordinary activity results in fatigue, palpitation, dyspnoea or anginal pain.
- Class III: Comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnoea or anginal pain.
- Class IV: Symptoms present at rest. All physical activity causes discomfort.



Some degree of mitral regurgitation is found in up to 30% of patients with coronary disease who are being considered for coronary bypass surgery. It is secondary to ischaemic damage to the papillary muscles, dilatation of the mitral ring or both. If mild or moderate the valve often becomes less regurgitant as the ventricle remodels after revascularisation. If severe, it is associated with a poor prognosis.

If the ejection fraction is less than 20% or the end systolic dimension greater than 5.0cms, valve surgery is generally not indicated as the added afterload on the ventricle after mitral competence is restored will likely be overwhelming for the damaged ventricle. In these patients, cardiac transplantation is recommended if the patient is under 65 years and diuretic and ACE inhibition therapy if over 65 years. An exception to this would be in patients with ischaemic valve disease in whom hibernating myocardium has been demonstrated by stress echocardiography or nuclear techniques.

Repair versus replacement

Although mitral valve replacement (with mechanical or bioprostheses) has been used successfully in the treatment of regurgitation since the early 1960s, there has been some dissatisfaction with the results of the operation. There is ample data demonstrating that the structural integrity of the mitral valve is essential for normal left ventricular function.

Whereas, in the past, the deterioration in left ventricular function was thought to be due to increase in afterload consequent on abolishment of the low impedance leak, it is now clear that the loss of annular-chordal-papillary continuity interferes with left ventricular function in those who have undergone replacement of the valve. This does not occur after mitral valve repair.

Thromboembolism or haemorrhage may occur after mechanical prosthesis placement and bioprostheses eventually fail from structural valve deterioration. Both suffer the hazard of infective endocarditis.

The mitral valve operation of choice depends on the physical characteristics of the valve. A heavily calcified annulus and/or valve requires replacement with a prosthetic or bioprosthetic valve. Generally disease of rheumatic aetiology warrants replacement. Pure or predominant regurgitation with pathology as ruptured chordae, prolapsed leaflets or annular dilatation should undergo repair of the mitral valve by standard repair techniques. Overall up to 50% of regurgitation is amenable to repair.

Calcification of the annulus is not a contraindication to repair as most calcium may be removed. However, calcification of the annulus is often associated with calcification of the leaflets, with thickening and tethering of the chordae making repair suboptimal. Repair of prolapse of the anterior leaflet is also more difficult.

Results

For degenerative valve disease, the operative mortality rate associated with mitral valve repair is lower than that with mitral valve replacement: early mortality rate ranges from 1-4% for patients undergoing repair versus 4-12% for replacement. There is enhanced systolic function after repair as opposed to replacement. Actuarial survival rates for repair are the range of 70-90% at 8-10 years and 70% at 15 years after repair. The estimate of freedom from reoperation for repair ranges from 93-95% at 8-15 years. Since fewer complications and a lower operative mortality rate are associated with mitral reconstruction compared with replacement, repair should be considered earlier in the natural history of the disease.

Ischaemic mitral regurgitation is associated with higher operative risk (9-30%). It is generally thought that repair is preferable to replacement when feasible as it can potentially reduce early complications and improve long-term survival. Cohn et al recently demonstrated that this worse postoperative prognosis was no better with mitral repair compared with replacement.

Summary of timing of surgery

- Mild degrees of regurgitation do not cause symptoms and do not require surgery.
- Moderate/severe regurgitation with symptoms warrants consideration of surgery, though severity of symptoms will dictate the need for an individual patient. The like-lihood of a reparable valve or, if recent atrial fibrillation has supervened, may justify surgery even if symptoms are minor.
- Severe regurgitation may produce no symptoms, but may lead to irreversible ventricular dysfunction which adversely affects long-term prognosis after surgery. Deterioration in systolic function, determined by ejection fraction or end systolic diameter (>4.5cms) indicates the need for valve surgery. If the regurgitation is secondary to myocardial ischaemia, the situation is less clear cut.
- If the patient is apparently asymptomatic with severe mitral regurgitation, it is worthwhile to closely question the patient to verify if there truly are no symptoms. It may be helpful to do an exercise test. If the results of this show an ejection fraction of >70%, end systolic dimension of <4cms, then it is reasonable to follow with echocardiography every 6 - 12 months.
- If the patient is aged >75 years, operation is indicated only for symptoms.

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