THE ROLE OF OPEN MITRAL VALVE REPAIR OR REPLACEMENT FOR SEVERE MITRAL STENOSIS OR REGURGITATION VERSUS CATHETER BASED THERAPIES

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INTRODUCTION

The development of various catheter-based therapies for valvular heart disease has provided less invasive, durable treatment options for certain selected groups of patients particularly young patients with uncomplicated rheumatic mitral stenosis (1,2). In these patients, results have been similar to those obtained with closed mitral commissurotomy operation (3,4). However, in many patients the extent and severity of pathological changes in the normal anatomy of the mitral valve still makes correction of these abnormalities under direct vision the best option.

ANATOMY AND PHYSIOLOGY

The mitral valve consists of an anterior and posterior leaflet attached to the heart at the mitral annulus. The leaflets are stabilized by chordae tendinae attached to the papillary muscles of the left ventricle. The annulus is poorly developed, consisting of the condensation of the posterior leaflet onto the left ventricular myocardium. It is important to note that the anterior and posterior leaflets are not separated at the anterolateral and posteromedial commissures. The commissural separation of the leaflets usually begins 3 to 8 millimeters from the annulus towards the mitral orifice. This appears to be important for maintenance of mitral competence.

During left ventricular systole the area of the mitral orifice is reduced by 23 to 40% by the effects of left atrial contraction and left ventricular contraction. This not only reduces the length of the posterior two-thirds of the annulus but also increases the height of the center of the antero-posterior dimension of the mitral annulus which is in the shape of a saddle. These changes in the annulus bring the posterior mitral leaflet into closer apposition with the anterior leaflet. Total leaflet area is about 150% of the area of the mitral orifice and the leaflets overlap by about one centimeter at the free edges during systolic apposition at their rough area.

The papillary muscles are located below the commissures of the mitral valve rather than the leaflets and account for up to 25% of left ventricular mass. Their anatomy is highly variable, ranging from the classical two well formed structures to no identifiable muscles at all. The annular papillary distance and alignment are critical for mitral competence.

PATHOLOGY OF MITRAL VALVE STENOSIS

The most common cause of mitral stenosis is rheumatic fever. Following the initial episode of rheumatic carditis, the inflamed leaflets adhere to each other on their atrial surfaces at the commissures, leaving a central orifice and causing mitral stenosis. In some cases this represents the main pathological change and the leaflets, chordae and papillary muscles remain otherwise relatively normal. These are the findings most commonly seen in young patients and in young patients sinus rhythm also may persist. This represents the ideal pathology for closed mitral commissurotomy or catheter based interventions because the leaflet fusion will usually separate along the original anatomic lines of the commissures and the chordae and papillary muscles will allow good leaflet motion. The resultant valve after commissurotomy is relatively normal.

Closed mitral commissurotomy has been a highly successful procedure in properly selected patients and the characteristics most favorable for this type

of surgery have been reported in analyses of large series of patients. These include young age, no calcification, normal leaflet motion especially of anterior leaflet, mobile subvalvular apparatus and minimal mitral regurgitation (5-8).

Unfortunately in many cases, especially in older patients or patients who have experienced severe carditis, changes in the leaflets, chordae tendinae, papillary muscles and left atrium are more severe and progressive. These valves have very severe fibrous leaflet fusion at the commisures which is often calcified and which will not separate without sharp dissection. The leaflets are thickened, shrunken in size and calcified, and are relatively immobile especially at the free edge. The chordae are shortened, thickened and fused together along with the papillary muscles. The chordae may almost disappear and the tip of the papillary muscle fuse with the leaflet. The atrial side of the orifice becomes deep and conical with a fixed stenotic orifice which also causes mitral regurgitation.

These types of valves cannot be treated successfully by closed methods. In some cases the valve destruction by fibrosis, calcification and ulceration is so extensive that prosthetic valve replacement is indicated. The left atrium enlarges over time with patchy fibrosis, hypertrophy and other cellular changes leading to chronic atrial fibrillation.

IMPLICATIONS FOR CHOICE OF THERAPY IN MITRAL STENOSIS.

The severity of the pathology of the stenotic mitral valve can be identified reasonably accurately by TEE. The grading system which examines leaflet mobility, valvular thickening, subvalvular thickening and valvular calcification on scales of 1-4 is useful (9). A number of studies have shown poorer outcomes in patients with valves graded at 7-8 or above (10). The average score in the randomized studies comparing closed commissurotomy with balloon valvotomy was 6.7 and age was 30 years (10). In patients with higher scores, radical open mitral commissurotomy techniques are required which may involve: sharp dissection of the commissures; splitting apart of papillary muscles and chordae; chordal division and synthetic PTFE chordal replacement; leaflet debridement, resection ± pericardial patch; ring and/or Kay annuloplasty (11-12).

When the severity of mitral regurgitation is already more than 2+, catheter techniques may be contraindicated because most catheter techniques have been shown to exacerbate mitral regurgitation in mitral stenosis in a significant proportion of patients by at least one grade (13,14). This is usually easy to correct when mitral commissurotomy is being treated by direct surgery. Acute, severe mitral regurgitation after catheter interventions usually can be corrected by open mitral valve repair.

PATHOLOGY OF MITRAL VALVE INSUFFICIENCY

In this country the two most common causes of mitral insufficiency are myxomatous degeneration of the mitral valve and acute or chronic ischemic cardiomyopathy. Myxomatous degeneration is characterized by enlarged, thickened prolapsing leaflets with elongated chordae and annular dilatation. This is thought to be due to structural deterioration of the leaflets and chordae secondary to abnormalities of fibrillin synthesis (15). Despite these changes, the majority of myxomatous valves are competent (16, 17). Mitral regurgitation develops because of severe annular dilatation and/or asymmetrical leaflet apposition due to chordal elongation or rupture. Most of these valves have a combination of problems.

The surgical techniques for treatment of myxomatous mitral regurgitation are well standardized with long-term follow-up of their effectiveness (18, 19), They involve direct reconstruction of the chordal and leaflet abnormalities combined with prosthetic ring annuloplasty. On average, ring annuloplasty reduces the annular diameter by 10-15mm. Long-term results are usually good. Elimination of all mitral regurgitation is routinely achieved.

The term ischemic mitral regurgitation describes a complex group of conditions usually associated with intrinsically normal valve mitral leaflets and chordae. Acute mitral regurgitation may occur after extensive inferior wall infarction with elongation of the annular-papillary muscle distance or with compromise of the posterior papillary muscle including papillary muscle rupture. Chronic ischemic mitral regurgitation is seen in association with inferior wall motion abnormalities causing a restrictive defect or in patients with diffusely poor left ventricular function with spherical dilation who have displacement of the bases of their papillary muscles downward and outwards, again causing restrictive

mitral regurgitation. Surgical correction involves marked reduction of the length of the posterior mitral annulus. This is despite the fact that the annular dimensions may be normal. The annuloplasty has been shown to bring the tips of the papillary muscles closer together and improves leaflet apposition (20-24).

INTERVENTIONS FOR MITRAL INSUFFICIENCY

The earliest efforts to correct mitral regurgitation prior to the availability of cardiopulmonary bypass involved attempts to displace the posterior annulus forward with foam plastic wedges and sutures. In most cases, however, the magnitude of reduction in annular dimensions required is so great that techniques utilizing the coronary sinus (usually located above the annulus) are unlikely to provide long-term benefit. The coronary sinus is also very delicate in elderly patients. However, in acutely ill patients with mitral regurgitation and low ejection fractions, even a modest reduction in mitral regurgitation may have a major beneficial impact and catheter based therapies may have an important role in these patients (25). In myxomatous disease, the Alfieri technique has been applied in which the center of the free edges of the leaflets are sutured together to produce a double outlet mitral orifice (26). Without the addition of annuloplasty to stabilize the repair, results have been poor.

MANAGEMENT OF ATRIAL FIBRILLATION

Atrial fibrillation develops eventually in mitral stenosis and regurgitation as the left atrium dilates and develops myocardial changes. Atrial fibrillation leads to embolic complications, impaired cardiac function and reduced long-term survival. Correction at the time of valve surgery is now performed in all our patients. The procedure of choice is the Maze procedure which has a 98% drug free cure rate of atrial fibrillation at three months of followup. Excision of the left atrial appendage is an integral part of the procedure.

CONCLUSION

Open isolated repair of stenotic or insufficient mitral valves is now usually performed through limited access exposures with a mortality of less than one percent. Open repair can be combined with other heart valve surgeries, coronary bypass or Maze procedures. Surgery and catheter based therapies should be viewed as complementary therapies.

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