Mitral Valve Regurgitation: Surgical Treatment

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Surgical treatment of mitral valve insufficiency aims at relief of symptoms, minimisation of complications and improvement of survival. The development of prosthetic mitral rings and the establishment of reconstruction methods have led to wide application of these techniques[1]. The ideal prosthetic valve has not yet been invented: late complications of both prosthetic mechanical valves (bleeding, thromboembolic events) and tissue valves (progressive failure) have contributed to a tendency to favour reconstruction whenever feasible[2]. By repairing a native valve, surgeons try to turn a diseased valve into a more normally functioning valve.

Historical data

Leonardo da Vinci described the anatomy of the mitral valve in the 15th century. L. Brunton initiated surgical treatment of the mitral valve, suggesting transventricular mitral dilation[3]. D. Harken demonstrated the value of digital commissurotomy in 1948. He predicted that the future of surgery for valvular disease would be associated with the direct imaging of intracardiac tissue and the development of extracorporeal circulation[4], which were achieved by W. Lillehei in 1957[5]. A few years later, in 1961, A. Starr successfully replaced a mitral valve with a prosthetic valve[6]. Major contributions were made by A. Carpentier, who developed tissue valves (1969), annuloplasty rings (1971) and most of the operative techniques for mitral valve reconstruction[7,8].

Anatomy and pathophysiology in mitral insufficiency

The decision for operation and the selection of prosthetic valve type or the kind of reconstruction should be based on the detailed study of the clinical manifestations, the laboratory findings and especially on the comprehension of the aetiology of the mitral insufficiency.

The mitral valve has six functional components: the leaflets (anterior and posterior), the chordae tendineae, the papillary muscles (anterolateral and posteromedial), the mitral annulus, the atrial myocardium and the left ventricular myocardium, which supports the papillary muscles and the annulus[9]. Any of these functional components, alone or in combination, can cause mitral regurgitation.

Mitral leaflets

The most common cause of acquired MR in North America is myxomatous degeneration, or "Barlow syndrome", or "floppy valve". The majority of patients are women. However, significant disease needing operative treatment is commonly seen in males over 50 years old. Patients with mild degenerative disease are usually not symptomatic or have mild symptoms. Nevertheless, significant mitral insufficiency is associated with high risk for sudden death, bacterial endocarditis and –in combination with atrial fibrillation– cerebral embolism[10]. In most cases the abnormality develops in the posterior leaflet or in both. The histological abnor-

mality is the replacement of part of the collagen of the leaflets with acid mucopolysaccharides. The chordae tendineae (thin, elongated or ruptured) and the annulus (dilated) are secondarily involved.

Acquired mitral stenosis is almost always due to rheumatic disease. In recent years there has been a slight increase in rheumatic fever in the USA[11]. Acute rheumatic fever causes inflammation and oedema of the leaflets and dilation of the annulus, while chronic disease leads to fibrosis with contraction and thickening of the leaflets, fusion of the leaflets at the commissures, fusion and contraction of the chordae tendineae and the papillary muscles. Valvular insufficiency and stenosis are usually both present, whereas insufficiency alone is seldom found in rheumatic heart disease[12].

Bacterial endocarditis is less common and may infect the leaflets, resulting in penetration, ulceration and contraction of the leaflets and thus causing MR. The most frequently found bacteria are streptococcus, staphylococcus aureus and Gram (-) bacteria[13].

Chordae tendineae

Automatic rupture is rather seldom and can be seen in younger patients, or during pregnancy, or after blunt trauma of the chest. Rupture of the chordae tendineae is usually secondary to mitral prolapse, Marfan syndrome, rheumatic heart disease, endocarditis etc.[14]

Papillary muscles

A common cause of MR is dysfunction of the papillary muscles, caused by ischaemic muscle damage or destruction of the adjacent ventricular wall. Arterial supply of the posteromedial papillary muscle is derived from the posterior descending coronary artery. Consequently, the posteromedial papillary muscle is affected more frequently by ischaemia than the anterolateral muscle, which has a double arterial supply (diagonal and marginal branches). Ischaemia and infarction of the papillary muscle can lead to muscle rupture, acute mitral insufficiency, acute heart failure and death. More often, however, fibrosis of the infracted area develops and MR is established gradually. During angina, the papillary muscle may temporarily dysfunction. In ischaemic cardiomyopathy the left ventricle and the mitral annulus are dilated[15].

The chordae tendineae and the papillary muscles (anterolateral and posteromedial) constitute the subvalvular apparatus**.** This apparatus makes a fundamental contribution to left ventricular function. Studies in patients who underwent mitral valve replacement with excision of the subvalvular apparatus, compared with patients with subvalvular apparatus preservation (after reconstruction or replacement), showed better results regarding operative mortality, postoperative function of the left ventricle and late survival for the second group[34,35]. Subvalvular structures support the left ventricle wall, minimise regional wall tension and promote ventricular contraction. Additionally, subvalvular structure preservation protects the left ventricle from postoperative rupture[22, 36].

Annulus

Dilation of the left ventricle of any aetiology is accompanied by mitral annulus dilation. It is of great importance to emphasize the dynamic nature of the mitral annulus: it has a large circumference at the beginning of atrial systole, but there is a decrease in annular area in the middle of the ventricular systole. This decrease plays an important role in tight leaflet sealing, thus avoiding mitral regurgitation[17]. Calcification of the annulus (10% in patients over 50 years of age) affects the dynamic nature of the structure and the motion of the leaflets, thus causing MR. Calcification may be primary (young females) or degenerative (diabetes mellitus, systemic hypertension, aortic stenosis)[18].

Indications for operation

Mitral valve repair is theoretically possible as long as the anterior leaflet is functioning. The cross-sectional area of the anterior leaflet alone is greater than the orifice area of most prosthetic valves. Extensive calcification or significant disease in the posterior leaflet is seldom a contraindication for reconstruction (this is possible if sufficient calcium can be safely removed from the posterior leaflet and if a ring annuloplasty is performed)[40].

Age is not a contraindication to valve repair. Patients 70 years of age or older have undergone mitral valve reconstruction with a hospital mortality rate of 4.4% and with a 5-year rate of freedom from cumulative cardiac death and re-operation of 74%[40].

Mitral reconstruction is the appropriate treatment in most patients with mitral insufficiency due to degenerative disease.

In rheumatic valves, the feasibility of repair varies on the kind of lesion (stenosis or insufficiency), the mobility of the leaflets, the condition of the subvalvular apparatus, etc. Although mitral repair is feasible for most patients with rheumatic disease, the 10-year rate of freedom from re-operation is low[41,49,77,80].

Mitral insufficiency secondary to bacterial endocarditis can be repaired with reconstruction if surgical repair is indicated and as long as the disease is discrete.

The presence of atrial fibrillation (use of anticoagulants) is not a contraindication for reconstruction, as long as a good repair result can be achieved. Development of atrial fibrillation in patients with MR is also an indication for operation. If the atrial fibrillation is of recent onset (<6 months) there is a high likelihood of sinus rhythm restoration after the operation[19,21,22].

Mitral insufficiency due to coronary artery disease is usually accompanied by ventricular dysfunction, so when significant stenosis of the coronary arteries is present surgical reconstruction should not be postponed[22,23].

Congenital insufficiency of the mitral valve is also an indication for surgical reconstruction.

Patients with multivalvular disease, severe rheumatic disease, severe thickening and calcification of the leaflets or of the annulus, should undergo replacement rather than reconstruction. Replacement should also be preferred in patients with severe renal insufficiency (accelerated calcification of the valve).

C. Atkins in Boston, having studied 263 patients with concomitant coronary artery disease and MR, claimed that severe insufficiency, low ejection fraction, acute myocardial infarction and acute heart failure are indications for valve replacement[50].

Patients with acute MR, secondary to post-infarction papillary rupture or chordae tendineae rupture, usually manifest cardiogenic shock. The only hope for these patients is emergency operation, although operative mortality approximates 50%. First of all, the cause of mitral insufficiency should be recognised, concomitant coronary artery disease should be estimated and the presence of post-infarction ventricular septal defect should be excluded[31,32].

Bacterial endocarditis can also be a cause of MR: there is a clear indication for emergency operation if, after more than one week's antibiotic treatment, the patient remains septic, there is haemodynamic deterioration and mobile vegetations can be seen on the echocardiogram[33].

In patients suffering from MR, the right timing of the move to operative treatment is essential for survival and for postoperative quality of life. In ideal conditions, the candidate for operation has significant MR with acceptable left ventricular ejection fraction and sinus rhythm.

Up until the late '80s, operation used to be postponed until symptoms of chronic MR became severe. This was due to the relatively high frequency of postoperative complications: increased 30 days mortality, deterioration in left ventricular function, thromboembolic events, bleeding, endocarditis, progressive malfunction of prosthetic valves, etc. The hazard of the previously mentioned complications exceeded the advantages from valve replacement in non-symptomatic or mildly symptomatic patients[19].

The increasing interest in MR valve reconstruction is due to the following reasons: a) postoperative complications of prosthetic valves; b) development of standard reconstruction techniques and annuloplasty rings; c) better late results compared to valve replacement; d) better intra-operative myocardial protection (repair takes less time than replacement); e) better systolic function of the left ventricle due to subvalvular structure preservation; f) increase of degenerative disease and decreased rheumatic disease[19,20].

Patients with severe MR, with symptoms affecting their quality of life, or patients with proof of severe myocardial dysfunction (by echogram or angiography) should be operated on as soon as possible[20].

In patients with severe left ventricular dysfunction, the differential diagnosis is between primary cardiomyopathy with secondary MR and primary MR with secondary cardiomyopathy. The operation is feasible even in patients with severe dysfunction of the left ventricle; these patients can be improved, though possibly only temporarily, if ejection fraction is at least 25% and severe pulmonary hypertension or renal and hepatic failure is absent. The aim of these palliative operations is the improvement of the patients' clinical condition, so that their symptoms may be better controlled with drugs[24,26].

The best timing for the operation of the patient without symptoms or with only mild symptoms depends on different indicators of the systolic function of the left ventricle: ejection fraction (EF), left ventricular end systolic diameter (L.V.E.S.D.), left ventricular end systolic volume index (L.V.E.S.V.I.), cardiopulmonary exercise stress testing with evalua-

tion of the maximum oxygen consumption (VO_2 max), relation of end systolic diameter with left ventricular wall thickness and size of the left atrium, regurgitated volume, etc.[19,25-29]

If L.V.E.S.D.I > 2.6 cm/m^2 and L.V.E.S.V.I >50 ml/m^2 systolic function of the left ventricle will remain severely impaired postoperatively[27,29]. On the other hand, L.V.E.S.D.I <2.5 cm/m^2, L.V.E.S.D. = 40-45 mm and L.V.E.S.V.I <50 ml/m^2 are good prognostic signs[27-,29].

Recently K. Fleischman has reported the importance of some clinical parameters that affect the postoperative result: age, N.Y.H.A. class, MR aetiology and calcification of the leaflets[30].

Patients without or with mild symptoms and satisfactory left ventricular function, should be followed up every 6-12 months; if a deterioration in left ventricular function is detected then an operation should be performed as soon as possible[19,22].

Surgical technique

In 1971, A. Carpentier published a functional classification of mitral valve disease. This classification is focused rather on surgical restoration of the valve physiology than on anatomic features of the insufficient valve. There are only 2 functional disorders: opening and closing of the valve is increased (leaflet prolapse) or decreased (leaflet restriction).

Type 1: valve with normal leaflet mobility. Regurgitation is due to annular dilation (heart failure) or leaflet perforation (endocarditis). **Type 2:** increased leaflet mobility. Regurgitation is secondary to leaflet prolapse, elongation or rupture of the chordae tendineae or papillary muscle (mitral prolapse, ischaemic disease). **Type 3:** restricted mobility of the leaflets. Insufficiency is caused by commissural coalescence, leaflet or chordae thickening (rheumatic disease)[8].

The preoperative or intraoperative echocardiogram has an essential role for the estimation of the pathology and the physiology of the mitral valve. Many surgeons, in addition, consider the direct view of the valve and the subvalvular structure to be an exact method of judging the lesion. The left atrium is dissected and, while the heart is still beating before cardioplegia, the pathology and the physiology of the valve are estimated[22].

The main reconstructive techniques are: a) posterior leaflet prolapse: quadrangular resection and sliding leaflet technique[1,40,41]; b) anterior leaflet prolapse: neochordae fashioned from PTFE suture, chordae transposition, chordal shortening, etc.[40,42,43]; c) restricted leaflet motion: resection of coalesced secondary chordae tendineae, commisurotomy, leaflet decalcification, subvalvular structure enlargement, etc.[1,79,80]; d) ischaemic regurgitation: rupture of chordae tendineae or papillary muscle is managed with muscle reimplantation, chordae transposition or replacement and mitral valve replacement. The reliability of these techniques depends on the emergency of the operation and the fragility of the valve tissue. Patients with previous myocardial infarction, akinesis or dyskinesis of the left ventricle and concomitant MR are better managed with prosthetic ring implantation, coronary artery bypass grafting, aneurysmectomy, transposition of dysfunctioning papillary muscles and / or valve replacement[22]; e) severe calcification of the mitral annulus**:** reconstruction demands experience and includes meticulous decalcification, segmental resection of the posterior leaflet and prosthetic ring implantation. More often, however, valve replacement is necessary[1,80,81].

Intraoperatively, the adequacy of mitral valve reconstruction is initially evaluated (while the heart is not beating and the left atrium is still open) by saline injected with force into the left ventricle, checking a residual valve leakage, and then (after the patient is weaned from cardiopulmonary bypass) by transoesophageal echocardiogram[22,82].

Annuloplasty rings

An ideal annuloplasty ring should be able to correct the abnormal dilatation of the posterior portion of the annulus, improve leaflet attachment, reinforce leaflet repairs and prevent further regurgitation, while restoring the normal annular circumference and the dynamics of the annulus (shape and size changes during the heart circle).

The basic prosthetic ring types are: a) Carpentier rigid ring, b) Duran flexible ring, c) Carpentier semi-flexible ring (Physio-ring), d) Cosgrove flexible partial ring (covers the posterior leaflet only). There are different results in many studies comparing rigid and flexible rings: a randomised clinical study (comparison between Carpentier rigid ring and Duran flexible ring) revealed better left ventricular function after 2-3 months when a flexible ring was used[37]. Echocardiographic studies showed a slight reduction in valve cross-sectional area without significant gradient when a ring is used, compared to recon-

struction without an annuloplasty ring. No difference was discovered between the two types of the prosthetic ring[38].

Although many surgeons do not recommend the use of an annuloplasty ring after mitral reconstruction, most of them believe that a ring strengthens the repair results and reduces regurgitation postoperatively. Of course, the implantation of a prosthetic ring is not without complications: partial abruption, systolic anterior motion (SAM) of the anterior leaflet leading to left ventricular outflow tract obstruction, haemolysis, etc[22].

When the mitral valve has to be replaced, a mechanical or a tissue valve can be used. Homografts or autografts (Ross II operation) have been used as well. Recently, C. Acar, presenting his experience from 102 cases of homograft implantation, emphasised that this technique cannot be used in all cases of mitral replacement and needs more clinical research[83].

Minimally invasive surgery

The principal of this kind of operation is to reduce the morbidity and the cost, to avoid blood transfusion, to speed hospital discharge and shorten the rehabilitation time. The mitral valve can be approached via a ministernotomy incision, right parasternal incision or right anterolateral mini-thoracotomy[68,69]. Cannulation is performed either directly through the incision or through femoral vessels (port access cardiopulmonary bypass system). The operation is performed under direct vision or is video assisted. Totally endoscopic robotic surgery is opening a new era in minimally invasive mitral valve surgery.

Disadvantages of the operations of that kind are complications affecting the central nervous system and ascending aorta during the learning curve (cerebral event, aortic dissection), ligation of the right internal thoracic artery, the high initial cost of the equipment needed and the long learning curve[67-69].

Good early and late results have been achieved with minimally invasive techniques compared to traditional techniques[67-70]. However, we should wait for the late results of multi-centre randomised studies to confirm the efficacy of these techniques[67].

Surgical treatment of atrial fibrillation

About 50% of the patients that undergo mitral valve operation have atrial fibrillation (Afib). Afib patients suffer systemic embolism twice or 3 times more frequently after valve replacement. Long lasting use of antiarrhythmic drugs can also create problems. Since 1991 the surgical treatment for atrial fibrillation is the maze procedure. The maze procedure usually accompanies mitral valve or coronary artery operations. It is a safe and effective treatment, with fewer late complications, in selected patients[71,72].

In the last 3 years radiation, microwaves, ultrasounds or cryoablation have frequently been used in the treatment of Afib. The advantage of these techniques is the safety for the patient and the minimal additional time needed. The basic disadvantage is the difficulty in achieving a transmural ulceration[73,74].

Although early results are very satisfactory and encouraging, we should wait for long term results and analysis of larger series.

Mitral regurgitation surgical results

Early results

During the last 15 years, in-hospital mortality after mitral valve reconstruction has been considerably improved, thanks to better reconstruction techniques, better intraoperative myocardial protection, and the tendency towards early surgical repair be fore severe ventricular dysfunction[22].

Early postoperative mortality depends considerably on the aetiology of the mitral regurgitation: it is 0-2% in patients with myxomatous degeneration, while it is 7-26% in patients with ischaemic disease[15,35,44-48]. In-hospital mortality is usually lower after reconstruction (1-4%) than after replacement (4-12%), mainly because of the preservation of the subvalvular apparatus[39,41,44,75,76]. Nevertheless, some patient subgroups with ischaemic disease have better survival after valve replacement[49]. Preservation of the subvalvular structure after mitral valve replacement contributes to better and longer preserved (7 years) systolic function of the left ventricle. Rarely, subvalvular structure preservation can lead to subvalvular stenosis or / and prosthetic valve dysfunction[22,34-36,53].

C. Atkins, after studying 263 patients with degenerative or ischaemic disease who underwent valve reconstruction (133 patients) or replacement (130 patients), concluded that early surgical mortality is influenced by age over 70 years, functional class, cardiac failure, emergency operation, replacement instead of repair, and rupture of the chordae tendineae of the anterior leaflet[50].

In-hospital mortality in patients with bacterial endocarditis is related with the kind (reconstruction: 2.6-29%, replacement 15.9-46%) and the emergency of the operation. Better results after reconstruction are due to the absence of prosthetic material, preservation of the function of the left ventricle and less severe disease[62,63].

Congenital lesions of the mitral valve are usually treated with reconstruction (93-95%) rather than replacement (5-7%). In-hospital mortality (4-21%) depends on the co-existence of other malformations, the infant's age, the severity of the regurgitation etc.[64,65.]

The use of a prosthetic ring is recommended by most authors (>87%)[39,41,76].

Latest results

The latest results support the use of the reconstruction techniques when feasible. In recent publications, the great majority of the patients (>95%) were in N.Y.H.A. class I or II after the operation[46,48,51,52], whereas most of them were in III or IV class before[2,46,48,51,52]. Patients who are in III or IV class soon after operation have a chance to improve their N.Y.H.A. class by the end of the first postoperative year[2,51,52].

Late survival after mitral valve surgery depends on the aetiology of the insufficiency, age, systolic function of the left ventricle, concomitant heart or non-heart diseases and the emergency of the operation.

Late survival after mitral valve replacement is reduced year by year, while it remains unchanged after valve reconstruction. This fact is due to the preservation of the subvalvular structure, thus maintaining good systolic function of the left ventricle, and on the absence of thromboembolic events, valve thrombosis and anticoagulant related haemorrhage[44,45,52].

After valve repair for degenerative disease, 5-year survival is 85-90%, 10-year survival 80%, and 15-year survival 70%[2,46,48,51,52]. As for rheumatic disease, survival is 90-96%, 84-93%, and 78%, respectively. The better results for rheumatic disease are related to the younger age of the patients[2,48].

Mitral regurgitation due to ischaemic disease is accompanied by worse early and late results. However, surgical treatment offers better results than medical treatment. C. Atkins has reported better 6-year survival and lack of cardiac complications after reconstruction (85%) than after replacement (73%) in patients with regurgitation of ischaemic aetiology[50,54]. Functional class of the left ventricle and aetiology of the regurgitation are the main prognostic factors for the results of the surgical treatment[49].

Prevention of infection (6 years follow-up) in patients operated due to endocarditis is better after reconstruction (95%) than after replacement (73%)[62].

Operations due to congenital mitral valve disease have better late (10 years) results after reconstruction (88% survival, 15% re-operation), than after replacement (51% survival, 28% re-operation)[65].

Thromboembolism: One of the most important advantages of mitral valve reconstruction compared to valve replacement is the relatively low thrombogenicity of the natural valve after the repair. Patients in sinus rhythm, without intracardiac thrombus preoperatively and without any other reason for anticoagulant therapy, do not need to take anticoagulants postoperatively. Permanent use of anticoagulants after repair is needed only in 30-50% of patients. Absence of thromboembolism following mitral valve reconstruction is 87-99% after 5 years, 81-94% after 10 years and 79-94% after 15 years[2,39,41,46,48,51,52,75,76]. The avoidance of anticoagulant use also eliminates the risk of bleeding[48,51,52].

Reoperations: Better late results after mitral valve reconstruction are due to the lower rates of reoperation. As far as degenerative disease is concerned, from one year after until the fifth year following the operation most patients are in good clinical condition (95-98%) and do not need reoperation. After 10 years their good condition rate is 88-95%, and after 15 years 85-90%[39,41,75,76].

In rheumatic disease, reoperation after one year is unnecessary in 95-98%, at 5 years 78-96%, at 10 years 84-94% and at 15 years 75-90%. The relatively high rate for late re-operation may be due to the progression of rheumatic disease[2,41,46,48,51,52].

Most reoperations take place during the first year after the operation and are due to residual regurgitation (technical error during the repair) or to an inexact intraoperative estimation of the repair result[45,47,52]. There is no doubt that reconstruction techniques have a learning curve. The need for reoperation at the end of the first year, particularly for degenerative disease, is rare: 1.3-8%[39,41,75,76]. Systolic anterior motion, leading to obstruction of the left ventricle outflow tract, is reported in up to 10% of some former reports[55,56]. However the gain of adequate surgical experience, in combination with a better understanding of the pathophysiology and the improvement of new surgical techniques, has led to a significant reduction in the rate of this complication (0-2.4%)[57,58].

Bacterial endocarditis: Absence of prosthetic material, preoperative antibiotics for active endocarditis, and prophylactic antibiotics perioperatively have led to obliteration of postoperative endocarditis in most reports of mitral reconstruction: 1-4% after 15-year follow up[2,39,41,46,52,59,75,76]. Bacterial endocarditis is also rare after valve replacement (1-9% after 5-year follow up), but is extremely difficult to treat[45,60,61].

Carpentier's team has recently reported late results from 434 patients who underwent mitral valve reconstruction during 1970-1984. Follow up was complete in 96% of the cases and the mean follow up time was 17 years (up to 29 years). Survival after 15, 20 and 25 years was 97%, 92% and 75% for rheumatic disease and 55%, 44% and 27% for degenerative disease, respectively. There was no need for reoperation in 70%, 52% and 40% for rheumatic disease and 93%, 93% and 93% for degenerative disease, respectively. Late results after repair are related more to the aetiology of the regurgitation than to the technique. Twenty years after reconstruction 48% of the patients with rheumatic disease had to undergo mitral valve replacement, compared to only 6.2% with degenerative disease. The remarkable results related to the treatment of degenerative disease must be due to complete repair of the lesions and the use of a prosthetic ring. Also remarkable are the low valve-related mortality rate and the absence of thromboembolic events, even for rheumatic disease[77].

Conclusions

Although mitral valve replacement is the treatment of choice for many clinical situations, the indications for repair are continuously being broadened. Carpentier reported a valve reconstruction rate of <5%, 25% and >75% during the '70s, the '80s and the '90s, respectively[78]. Better long-term results (10 years) after reconstruction are related to early surgical treatment, preservation of the left ventricle's systolic function and the prevention of prosthetic valve complications, while worse results are due to surgical technique, progression of the disease and concomitant heart disease (aortic valve disease, coronary artery disease).

The feasibility of the repair of mitral insufficiency depends on the pathogenesis of the regurgitation, the patients' willingness, the experience of the surgeon, the hospital facilities (transoesophageal ultrasonography, cost, waiting list), as well as on the right preoperative cardiologic evaluation.

For asymptomatic patients it is worth mentioning Carpentier's apophthegm: "These patients may have an uncomplicated life for many years. Consequently, we must be able to guarantee a successful valve repair before we lead them to the operating room".

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