MITRAL VALVE REPLACEMENT WITH CHORDAL PRESERVATION

A RETROSPECTIVE ANALYSIS OF OUTCOME IN COMPARISON WITH CLASSICAL MITRAL VALVE REPLACEMENT

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Certificate

     *This is to certify that the dissertation entitled "**Mitral valve replacement with chordal preservation a retrospective analysis of out come in comparison with classical mitral valve replacement**" presented here is the original work done by **Dr.K.Sakthivel** in the Department of Cardio Thoracic Surgery, Government General Hospital ,Madras Medical College, Chennai 600003, in partial fulfillment of the University rules and regulations for the award of M.Ch Cardiothoracic degree under our guidance and supervision during the academic period from 2006 to 2009.*

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CONTENT

Introduction

Rheumatic heart disease plays a major debilitating role in Indian population causing high morbidity and mortality, affecting the population in their productive phase of life. It impairs the quality of life and leads to psycho-social and economic burden to their family and the society. With the clinical introduction of cardiac valvular prosthesis in 1960, valve replacement has saved a number of lives affected by hemodynamically significant valvular heart diseases.

Prosthetic valve replacement is not with out danger and it became evident that "valve replacement is simply exchanging one disease for another". In spite of tremendous strides toward perfecting materials and design, the ideal valve remains elusive. Thromboembolism continues to be a major cause of morbidity and mortality in patients who have undergone mechanical valve replacement.

Recent surgical trend in treating rheumatic mitral valve disease is to repair the valve. If repair is not feasible, then valve replacement is still undertaken knowing the complications unique to prosthetic valve replacement in the interest of saving the life.

It is well known that traditional mitral valve replacement when compared to mitral valve repair carries a higher morbidity and mortality. This has been attributed to the preservation of the mitral subvalvular apparatus in repair techniques, but not all valves can be repaired, especially those of rheumatic etiology. Mitral valve replacement has been the procedure of choice usually adopted in these conditions, but the results have not been comparable. By preserving the annular ventricular continuity in mitral valve repair good left ventricle (LV) function in both early and late post operative period has been achieved.

A surgery in which annular-papillary continuity is preserved during replacement has shown better early and late results than traditional mitral valve replacement (MVR) techniques. As the awareness of the deleterious effects of the loss of annuloventricular continuity has increased chordal preservation during mitral valve replacement has gained in popularity.

ANATOMY OF MITRAL VALVE

The mitral valve apparatus consists of leaflets, annulus, chordae tendinae, papillary muscles, and left ventricle. Mitral valve and its apparatus play an important role in maintaining the left ventricular geometry and function.

ANNULUS:-

The mitral annulus is the area of leaflet attachment to muscular fibers of the atrium and ventricle. The annulus is pliable, permitting sphincteric contraction and decreases in diameter during each systolic contraction by approximately 26%. The orifice of the mitral valve also changes shape, from elliptical during ventricular systole to circular during late diastole. This flexibility increases leaflet coaptation during systole and maximizes orifice area during diastole.

Changes in size and shape of the annulus result from relaxation and contraction of the basoconstrictor muscles (bulbospiral and sinospiral bundles). In the horizontal plane the annulus is saddle-shaped. Anteriorly, the annulus is attached to the fibrous skeleton of the heart, extending between the two fibrous trigones. This limits its pliability and its capacity to dilate with mitral regurgitation (MR). The posterior annulus is more flexible and is not attached to rigid surrounding structures. This is confirmed by the clinical observation that dilation of the annulus occurs posteriorly with MR.

Anatomical knowledge of the mitral annulus and surrounding structures is critical to avoid inadvertent damage during mitral surgery. The circumflex coronary artery runs laterally around the mitral annulus in the posterior atrioventricular groove. The coronary sinus runs more medially in the same groove. The artery to the atrioventricular node, usually a branch of the right coronary artery, runs parallel and close to the annulus of the anterior leaflet near the posteromedial commissure. The aortic valve is situated between the anterior and posterior fibrous trigones. The bundle of His is located near the posterior trigone.

ANNULAR SIZE, SHAPE AND DYNAMICS

The average mitral annulus cross-sectional area ranges from 5.0 to 11.4 cm^{2}in normal human hearts (average is 7.6 cm^2). The posterior annulus circumscribes about two thirds of the mitral annulus. The magnitude of change in mitral annular area is in the 20% to 40% range. Annular area decreases to a minimum in early to mid systole.

The human mitral annulus is roughly elliptical (or D- or kidney-shaped), with greater eccentricity (i.e., being less circular) in systole than in diastole, in its most elliptical configuration, the ratio of minor to major diameters is approximately 0.75. During the cardiac cycle, annular regions adjacent to the posterior leaflet (where the leaflet attaches directly to the atrial and ventricular endocardium) move toward (during systole) and away from (during diastole) the relatively immobile anterior annulus

LEAFLETS:-

The mitral valve has two leaflets, the anterior (aortic) and posterior (mural) leaflets. The leaflets are attached to the mitral annulus and at its free border to the papillary muscles by primary and secondary chordae. The anterior mitral leaflet is in direct continuity with the fibrous skeleton of the heart. This leaflet is contiguous with the left and noncoronary cusps of the aortic valve and the area below the intervening aortic commissure, termed the fibrous subaortic curtain. Although the anterior leaflet

occupies only 35% to 45% of the annular circumference, its leaflet area is almost identical to that of the posterior leaflet.

The posterior leaflet is rectangular. The free margin of the posterior leaflet has two clefts that divide the posterior leaflet into three scallops: the largest or middle scallop, the posteromedial scallop, and the anterolateral scallop. Fan-shaped chordae insert into and define the clefts between the individual posterior scallops. Motion of the posterior leaflet is more restricted than that of the anterior leaflet; however, both mitral leaflets contribute importantly to effective valve closure.

The valvular surface area is bigger compared to the orifice area. The ratio is close to 2:1. This difference exaggerates even more during systole because of the sphincter action of annulus which reduces further the orifice area.

There are three phases in valve orifice closure.
1)   Edge to edge meeting between leaflets.
2)   Upward bulge of leaflet.
        3)   Final phase maximal coaptation of leaflet. Almost they lie vertically.

The surface of the mitral leaflet is divided into three zones corresponding to areas of chordal insertion and leaflet coaptation. The rough zone is the leading edge of the anterior and posterior mitral leaflets. This zone is the contact surface of the mitral leaflets during systole. The clear zone is peripheral to the rough zone and represents most of the body of the leaflet; this portion of the mitral valve billows into the atrium during ventricular contraction. The basal zone, between the clear zone and the annulus, receives the insertion of the basal chordae tendinae (tertiary chordae), which originate directly from the trabeculae of the left ventricle. The basal zone is found only on the posterior leaflet.

The histological structure of the leaflets includes three layers: (1) the fibrosa, the solid collagenous core that is continuous with the chordae tendinae; (2) the spongiosa, which covers the atrial aspect and forms the leaflet leading edge (it consists of few collagen fibers but has abundant proteoglycans, elastin, and mixed connective tissue cells); and (3) a thin fibro elastic covering of most of the leaflets. On the atrial aspect of both leaflets, this surface (the *atrialis*) is rich in elastin. The ventricular side of the fibroelastic cover (the *ventricularis*) is much thicker; it is confined mostly to the anterior leaflet and is densely packed with elastin.

CHORDAE TENDINAE:-

The chordae tendinae are chords of fibrous tissue that attach the mitral leaflets to either the papillary muscles or the left ventricular free wall. They often subdivide and interconnect before they attach to the leaflets. The chordae are divided into primary, secondary, and tertiary chordae.

Primary chordae attach directly to the fibrous band running along the free edge of the leaflets. These chordae ensure that the contact surfaces (rough zone) of the leaflets coapt without leaflet prolapse or flail. Secondary chordae attach to the ventricular surface of the leaflets at the junction between the rough and clear zones. These chordae contribute to ventricular function. Secondary chordae helps the ventricle to contract in an efficient cone-shaped fashion; when secondary chordae are excised, the left ventricle assumes a globular shape. Tertiary chordae are unique to the posterior leaflet. They arise as strands directly from the left ventricular wall or from small trabeculae to insert into the ventricular surface of the leaflet near the annulus.

PAPILLARY MUSCLES:-

The anterolateral and posteromedial papillary muscles each supply chordae tendinae to both leaflets. The two groups of papillary muscles support the anterolateral and posteromedial commissures. It arises from the junction of the apical and middle thirds of the ventricular wall.

The anterolateral papillary muscle receives a dual blood supply from the anterior descending coronary artery and either a diagonal branch or a marginal branch of the left circumflex artery. The posteromedial papillary muscle receives its blood supply from either the left circumflex artery or a distal branch of the right coronary artery.

Because of the single blood supply to the posteromedial papillary muscle, infarction of the posteromedial papillary muscle is much more common. Blood supply comes through a large central artery at its base from epicardial vessel then dividing in to network to supply the papillary muscle. The occlusion of large central artery severely damages the entire papillary muscle, due to lack of collateral blood supply. The occlusion of central artery causes necrosis of the papillary muscle which indeed leads to mitral regurgitation.

LEFT VENTRICLE:-

The posterior left ventricular wall and papillary muscles play an important role in leaflet coaptation and valve competence. Papillary muscles are aligned parallel to the ventricular wall and attach via chordae to the free edges of the valve leaflets. These muscles project from the trabeculae and may be single, bifid, or a row of muscles arising from the ventricular wall. During isovolumetric contraction the mitral leaflets are pulled downward and together by this interaction.

Ventricular dilatation may affect the alignment and tension on the papillary muscles and valve competence.

ATRIAL FIBRILLATION (AF):-

This is the commonest rhythm disturbance that accompanies mitral valve disease. In Atrial fibrillation walls of the atria shudder fast and atrial contraction will not be present. At the same time ventricular rate is fast and irregular due to chaotic bombardment of atrioventricular (AV) node by more than 700 impulses per minute from the atria.

The trigger of an AF is a focus of acute localized wall stretch. Occurrence of AF has been consistently related to the size of the left atrium. Univariate analysis in one study revealed that the incidence of AF was 3% when the left atrial diameter was less than 4.0 cm but increased to 54% if the left atrial diameter was more than 4.0 cm, thus explaining the highest incidence in patients with mitral valve disease, which causes maximum left atrial enlargement. For AF patients, sinoatrial (SA) node histology indicates severe degeneration of normal pacemaker tissue.

It is postulated that fibrosis and degeneration of the atrial myocardium in valvular heart disease, especially of rheumatic etiology disturbs impulse propagation in the atria and leads to AF.

Atrial fibrosis probably contributes to persistent AF after valvuloplasty or valve replacement/repair. In patients with valvular disease, AF also occurs more frequently with mitral valve calcification, mitral valve prolapse and following valve replacement surgery.

Atrial wall shows myofibrillar changes with increased fibrous tissue deposition, leading to haphazard electrical activity. Mechanically there is a loss of atrial contribution to stroke volume by up to 30%.

Combination of loss of atrial kick and fast ventricular rate (loss of diastolic filling time) occur plus loss of normal presystolic closure timing of atrioventricular valves due to fast ventricular rate and absent atrial contraction, all of which decreases cardiac output and mitral regurgitation may occur.

Dilatation of atrial wall leads to increased secretion of atrial natriuretic peptide. Fast heart rate precipitates congestive cardiac failure by tachycardia mediated cardiomyopathy there by AF per se is responsible for overt heart failure by increasing myocardial metabolism by provoking rapid myocardial contraction leading to exhaustion.

Dilated atrium, loss of atrial contraction is factor for clot formation. After the onset of AF, stroke is the

most feared and calamitous complication. Compared to sinus rhythm AF carries an increased mortality of about 1.5- 1.9 independent of heart disease and age and part of this increased mortality is likely due to stroke in its various presentation.

EVIDENCE BASED TREATMENT

The Committee on Management of Patients with Valvular Disease was given the task of reviewing and compiling this information base and making recommendations for diagnostic testing, treatment and physical activity. These guidelines follow the format established in previous American College of Cardiology / American Heart Association (ACC/AHA) guidelines for classifying indications for diagnostic and therapeutic procedures:

Class I: Conditions for which there is evidence and / or general agreement that a given procedure or treatment is useful and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

IIa: Weight of evidence/opinion is in favor or usefulness/ efficacy

IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful and in some cases may be harmful.

INDICATION FOR MITRAL VALVE REPLACEMENT

Indications for valve replacement pertain to those types of valve that are unlikely to be repaired or which have been shown to have poor long-term success after previous intervention.

MITRAL STENOSIS (MS)

Mitral stenosis is almost exclusively caused by rheumatic fever. The pathologic changes in rheumatic valvulitis are mainly fusion of the valve leaflets at the commissures; shortening and fusion of the chordae tendinae; and thickening of the leaflets due to fibrosis, contraction and calcification.
Stenosis usually develops one or two decades after the acute illness of rheumatic fever with no or slow onset of symptoms until the stenosis becomes more severe. Limitation of exercise tolerance is usually the first symptom followed by dyspnea that can progress to pulmonary edema. New onset atrial fibrillation and risk for thromboembolism, hemoptysis, and pulmonary hypertension are other common symptoms in patients with mitral stenosis.
The diagnostic workup of the symptomatic patient with mitral stenosis should include a complete cardiac catheterization, including coronary angiography in any patient over the age of 40. Under the age of 40, echocardiography findings of the mitral valve is enough in most symptomatic patients for the definition of mitral valve pathology unless there is a history of chest pain or coronary artery disease.
In general, operation is prescribed when the mean valve area is 1.0 cm^2 or less (normal mitral valve area: 4–6 cm^2 - however, with a "mixed" lesion of mitral stenosis and mitral regurgitation, the valve area in symptomatic patients occasionally may be as large as 1.5 cm^2. Asymptomatic patients are generally not considered for surgery but it is in the asymptomatic patients with significant hemodynamic mitral stensosis, still surgery is considered.

ACC / AHA RECOMMENDATIONS FOR MITRAL VALVE REPLACEMENT IN MITRAL STENOSIS

1. Patients with moderate or severe mitral stenosis (mitral valve area ≤1.5cm^2) and NYHA functional Class III-IV symptoms who are not considered candidates for percutaneous balloon valvotomy or mitral valve repair – Class I
2. Patients with severe mitral stenosis (mitral valve area ≤ 1 cm^2) and severe pulmonary hypertension (pulmonary artery systolic pressure > 60 to 80mm Hg) with NYHA (New York Heart Association) functional Class I-II symptoms who are not considered candidates for percutaneous balloon valvotomy or mitral valve repair – Class IIa.

MITRAL REGURGITATION (MR)

The etiology of mitral regurgitation is very diverse. Except in cases of acute ischemic mitral regurgitation and endocarditis, where indications are more straightforward. Indication generally for regurgitative lesion is complex. Etiology of regurgitation are degenerative (mitral prolapse, ruptured/elongated chordae), rheumatic, infectious and ischemic diseases of the mitral valve. Most of these are now amenable to mitral valve repair and reconstruction.

For any of the preceding major pathologic subsets, indications for surgery in patients with mitral regurgitation vary from the asymptomatic patient with an enlarging but well functioning left ventricle and atrium to severely depressed left ventricular function. Any symptomatic patient with significant mitral regurgitation (3+ to 4+) should be operated on, and operation should be considered in any relatively symptom free individual if there is objective evidence of left ventricular deterioration and significant increase in left ventricular end-systolic and end-diastolic volumes.
Regurgitation through the valve is usually measured with Doppler echocardiography, but magnetic resonance imaging (MRI) is another noninvasive technology for measuring the regurgitant flow and can provide measurements of ventricular end-diastolic/systolic volumes and ventricular mass. Left ventricular angiography with coronary angiogram can be helpful but is otherwise indicated for evaluating the coronary arteries preoperatively in patients older than 40 years.

It is important to stress that ejection fraction is a poor indicator of left ventricular function in patients with mitral regurgitation. Depressed cardiac output (< 40%) therefore usually indicates severe left ventricular dysfunction, and results of surgery are not as favorable in these patients as they are in patients with normal ventricles. Compared to ejection fraction, measurements of end-systolic volume and diameter are more reliable noninvasive parameters to evaluate the status of the left ventricle and determine the optimal time for operation.
 Carpentier et al classified mitral regurgitation into three pathoanatomic types based on leaflet and chordal motion: normal leaflet motion (type I), leaflet prolapse or excessive motion (type II), and restricted leaflet motion (type III). Type III is further subdivided into "a" and "b" based on leaflet restriction during diastole (type IIIa) or during systole (type IIIb), as typically seen in patients with ischemic mitral regurgitation (IMR).
Mitral regurgitation with normal leaflet motion is caused by annular dilation, which is often secondary to left ventricular dilation; as a rule, insufficient leaflet coaptation area or incomplete mitral leaflet coaptation is present. Examples include patients with dilated cardiomyopathy and some with ischemic heart disease complicated by ischemic mitral regurgitation (IMR). Normal leaflet motion is also

associated with leaflet perforation secondary to endocarditis.

Leaflet prolapse typically results from a floppy mitral valve with chordal elongation and/or rupture, but can be seen in patients with coronary artery disease who have papillary muscle rupture or rarely papillary muscle elongation. Mitral regurgitation due to restricted leaflet motion is associated with rheumatic valve disease (type IIIa and type IIIb), ischemic heart disease (IMR with type IIIb restricted systolic leaflet motion with or without annular dilation), and dilated cardiomyopathy (type IIIb plus annular dilation).

In chronic mitral regurgitation the left ventricular function gradually declines. The regurgitant volume into the left atrium during systole is added to the forward stroke volume and tends to increase the total forward output and ejection fraction in early phase. However progressive left ventricular dilation increasing the wall tension which leads to increased systolic wall stress and afterload. After mitral valve replacement, with rapid rise in the afterload, adaptation of the left ventricle to this change is based on the annulo ventricular continuity.

Fixation of the mitral annulus with a rigid prosthesis interferes with the distention and contraction of the basoconstrictor. Also after mitral valve replacement the left ventricular volume decreases because of the elimination of the regurgitant left atrium (LA) volume. After mitral valve replacement with chordal transection the ejection fraction (EF) is determined by contractility; preload and after load. Afterload is increased because of loss of low impedance pathway into LA; also reducing preload is the loss of regurgitant LA volume plus a residual gradient across the prosthesis impedes left ventricular filling.

All this contributes to a low cardiac output syndrome despite satisfactory prosthetic valve function. All the above mentioned problems were not seen with traditional mitral valve repair technique.

ACC/AHA RECOMMENDATIONS FOR MITRAL VALVE SURGERY IN NONISCHEMIC SEVERE MITRAL REGURGITATION

1. Acute symptomatic mitral regurgitation in which repair is likely – Class I.

2. Patients with NYHA functional Class I, III, or IV symptoms with normal left ventricular function defined as ejection fraction > 0.60 and left ventricle end-systolic dimension (LVeSD) < 45 mm – Class I.

3. Symptomatic or asymptomatic patients with mild left ventricular dyfunction, ejection fraction 0.50 to 0.60, and end-systolic dimension 45 to 50 mm – Class I.

4. Symptomatic or asymptomatic patients with moderate left ventricular dysfunction, ejection fraction 0.30 to 0.50, and / or end-systolic dimension 50 to 55 mm – Class 1.

5. Asymptomatic patients with preserved left ventricular function and atrial fibrillation – Class IIa.

6. Asymptomatic patients with preserved left ventricular function and pulmonary hypertension (pulmonary artery systolic pressure > 50mm Hg at rest or > 60 mm Hg with exercise) – Class IIa

7.   Asymptomatic patients with EF 0.50 to 0.60 and left ventricle end-systolic dimension <45 mm and asymptomatic patients with ejection fraction > 0.60 and left ventricle end-systolic dimension 45 to 55 mm – Class IIa.

8.   Patients with severe left ventricular dysfunction (ejection fraction < 0.30 and / or end-systolic dimension > 55 mm) in whom chordal preservation is highly likely – Class IIa.

9.   Asymptomatic patients with chronic MR with preserved left ventricular function in whom mitral valve repair is highly like – Class IIb.

10.   Patients with mitral valve prolapse and preserved left ventricular function who have recurrent ventricular arrhythmias despite medical therapy – Class IIb.

11.   Asymptomatic patients with preserved left ventricular function in whom significant doubt about the feasibility of repair exists – Class III.

REVIEW OF LITERATURE

Cardiac valve repair or replacement under direct vision awaited the development of the heart-lung machine. With the contribution by John Gibbon to the development of the heart-lung machine, valve replacement becomes feasible.

The first successful prosthetic mitral valve replacement was done by Nina Braunwald in 1960 [1]. This was followed by Starr and Edwards, replacing the mitral valve with a caged ball valve in September 1960, from then problems of post operative low cardiac output, thromboembolism, anticoagulation related bleeding came to the fore [2].

The first mitral valve prosthesis was implanted on Sept 21 1961 by Albert Starr and Edwards from University of Orgean. Problems of post op low cardiac output, thromboembolism, anticoagulation and bleeding came to the fore [3].

Central flow design offers a lower transvalvular gradient, durable, thromboresistance, low incidence of valve related adverse events. Of the more than 70 mechanical valves in clinical usage the St Jude medical valve has been the most successful and has been the gold standard for mitral valve replacement [4, 5, 6, 7, 8] in all age groups, from infant to the elderly [9,10,11,12,13,14].

According to Crawford etal the immediate operative mortality for valve replacement may not be related to the type of prosthesis used [15].

Lim etal reported from their study that Carbomedics and St Jude valve had no significant difference in early and midterm clinical outcomes. The choice with respect to valve type can be based on other than patient outcomes [16].

Ye-Ying Cen etal in their analysis of Carpentier-Edwards bio prosthesis and St Jude mechanical valve found, 10-year survival was not statistically different between the two but factors predicting adverse survival after mitral valve replacement were older age, lower ejection fraction, presence of class IV congestive heart failure, coronary artery disease, renal disease, smoking history, hypertension, combined valve surgery, and redo heart surgery. Choice of biologic or mechanical prosthesis does not significantly affect long-term patient survival after mitral valve replacement [17].

According to Ikonomidis etal valve replacement patients do not survive in parallel to normal population. Factors influencing the operative mortality are age, previous valve replacement, cardiac function, coronary artery disease. These factors influence on operative mortality than the type of valve prosthesis implanted. Late death is unrelated to prosthetic valve, and the factors influencing the long term outcome is higher NYHA (New York Heart Association) class, concomitant CABG, increasing age [5].

Remadi etal in their study identified age and sex as significant risk factors for valve-related mortality and that higher age, female sex, higher NYHA class, and atrial fibrillation had significant correlation with overall higher mortality [6].

In 1922 Wiggers and Kats and later Rushmer et al proposed the concept of annulo -ventricular continuity. According to this concept, the LV geometry and function are a result of a dynamic interaction between mitral annulus and left ventricular wall. The attachment between the mitral annulus and LV wall moderates the LV distensibility during diastole and wall stress during systole [18].

In 1960 McGoon and colleagues described an effective repair for MR due to ruptured chordae [19].

In 1964 Lillehei introduced the concept of chordal preservation during mitral valve replacement to reduce the problems of post operative low cardiac output syndrome. But his observations went unheeded at that point of time [20, 21].

In the original technique described by Lillehei the posterior leaflet was bound to the annulus with a running stitch. He reported a reduction of peri operative, mortality from 37% to 14% [20].

During the same period Mitral valve repair gained acceptance by removing the need for anticoagulation and better left ventricular function and hemodynamics afforded by it.

In1981 David reintroduced the concept of annulo ventricular continuity after extensive experiments in canine models with mitral valve replacement. He introduced the concept of total chordal preservation[22, 23, 24].

In the technique originally described by him the anterior mitral leaflet (AML) is incised at its base and carried to both sides and brought centrally towards the free edge of the leaflets and a triangular segment of the leaflet is excised leaving the chordae attached to the free edge which is resuspended to the mitral annulus by sutures used to secure the prosthetic valve.

The posterior mitral leaflet with its chordae is left intact, in patients with myxomatous mitral regurgitation undergoing mitral valve replacement. Chordae are shortened by imbricating the posterior mitral leaflet in the mitral annulus using sutures used for mitral valve (MV) fixation. The advantage he claimed with this technique was that the chordae were maintained in their natural anatomic location with reduced risk of left ventricular outflow tract obstruction (LVOTO) with the reduction in the bulk of leaflet tissues.

Miki Goor and Hennin and their colleague's clinical result suggested that chordal preserving during mitral valve replacement improved hospital survival and global left ventricular function. Carabello's group again documented preservation of systolic ejection performance in mitral valve replacement with chordal preservation.

In 1985 Feike et al reported a technique where the anterior leaflet was split from the centre of the free edge to the annulus. Incisions were made on either side of the split towards the commisures to detach the anterior mitral leaflet from the annulus.

The remaining two halves of the leaflet were left with the intact chordae. Leaflet was trimmed to remove thickened and calcific areas and then rotated posteriorly and sutured to the posterior mitral annulus. This technique was suitable in implanting tilting disc prosthesis where disc entrapment by the subvalvar apparatus was of concern[25].

Disadvantages reported were that this disturbs the normal geometry and relationships of mitral subvalvar apparatus which could alter the distribution of the left ventricular wall stress, disturb chordal tension during papillary muscle contraction thereby reducing the global left ventricular systolic and diastolic function.

In 1988 Khonsari et al described a technique of total chordal preservation where after detaching the annulus between the two commisures an ellipse of tissue was excised and the rim of leaflet tissue containing chordae was attached to the annulus (Khonsari I). If leaflet was thickened and calcified it was then divided into 2-5 segments bearing chordae which were re attached to the annulus (Khonsari II). Posterior mitral leaflet was retained in toto. Using this technique no left ventricular outflow tract obstruction or prosthetic function impairment was claimed[26].

Myocardial rupture was prevented by maintaining the tethering effect of the intact sub valvar apparatus.

In1988 Miki's described a technique to maintain more normal chordal tension wherein anterior mitral leaflet was incised and separated from annulus and divided at its center, anterior and posterior commisures were incised and papillary muscles were slit, excessive cuspal tissue and fibrous calcific nodules were excised. The two chordal segments thus created were sutured to the respective antero lateral and posteromedial commisures.

Posterior mitral leaflet was incised in center and prosthetic valve seated in position plicating the posterior mitral leaflet and including the posterior mitral leaflet and chordae in the sutures for securing valve. Advantages claimed were that this technique was simple, there is no left ventricular outflow tract obstruction, allows a larger prosthesis to be seated and very well suited for low profile valves[27].

In 1990 Rose and Oz described a technique were the anterior mitral leaflet was stretched posteriorly and a central portion was excised, the rim of the remaining leaflet tissue containing the marginal

chordae, defect in the anterior mitral leaflet was closed with a running suture parallel to the annulus. Valve sutures reinforced the previous suture line. Prosthetic valve was sutured to orient the leaflets perpendicular to the native orientation[28].

There were low chances of left ventricular outflow tract obstruction with reduced bulk of leaflet tissue, reduced risk of thrombosis on the residual leaflet[29, 30].

Vander Slam described yet another technique for preserving anterior leaflet[31].

Sarris et al demonstrated that in an open chest swine model the changes induced by chordal transection could be completely reversed by reattaching papillary muscle to the mitral annulus[32, 33].

Experiments by Hansen and associates demonstrated that left ventricular function was superior with intact subvalvar apparatus, intermediate with preservation of posterior mitral leaflet alone and poorest with loss of all chordae[33].

Horskote et al showed that partial chordal preservation preserving posterior mitral leaflet alone improved event free survival[34].

Hennein et al showed that after chordal excision, exercise capacity, left ventricle end-systolic dimension and cardiac index did not improve and that the left ventricular function declined. In contrast after preservation of the entire sub valvar apparatus the exercise capacity improved markedly, left ventricular function improved and early ejection fraction improved [22]

David et al reported results in patients undergoing redo mitral valve replacement after initial mitral valve replacement, where all chordae were transected. 4-0 PTFE (poly tetra fluro ethylene) sutures were used to create new chordae if they were transected previously. Incidence of low cardiac out put and operative mortality was low in the chordal preservation and reconstruction group[36].

Hetzers study reported major advantages of chordal preservation in the form of reduction in the mortality, improved early and late left ventricular function, improved long term survival and elimination of the risk of the dreaded complication of midventricular rupture[37].

Hassan Mottcha et al in their study noted that total chordal preservation did not cause left ventricular tract obstruction after mitral valve replacement[38].

Ghiskine Deklunder et al in their prospective study noted that anterior chordal transaction impairs not only regional left ventricular function but also regional right ventricular function in mitral regurgitation.[39]

AIMS AND OBJECTIVES

The aim in this study was to study in retrospect the possible out come benefits of chordal sparing surgery when mitral valve replacement is done for rheumatic mitral disease in terms of left ventricular function by assessing parameters of left ventricular systolic and diastolic function.

The parameters assessed were hemodynamic stability in the immediate peri and post operative period, left ventricular ejection fraction preoperative and postoperative, left ventricular end systolic and diastolic dimensions pre and postoperative .

MATERIALS AND METHODS

All patients who underwent mitral valve replacement between January 2008 and December 2008 for isolated mitral valve disease are studied. Patients with other valvular (aortic, tricuspid & pulmonary) or congenital cardiac defects requiring additional intervention were excluded from the study.

One hundred and fifty two patients underwent mitral valve replacement surgery for chronic mitral valve disease, in 86 of these procedures the classical mitral valve replacement technique was followed and in 66 patients chordal sparing technique was followed.

In both group of patient's preoperative NYHA class, left ventricular end systolic, end diastolic dimensions and preoperative ejection fraction were noted. Surgery was conducted with a standard midline sternotomy incision, bicaval and aortic cannulation. Core was cooled to 28°C.

Heart was arrested with hyperkalemic blood cardioplegia, with topical ice slush being used to cool myocardial temperature further. Left atrium was opened parallel to the interatrial groove. Surgery was conducted after inspecting the valve and suitability for chordal preservation assessed.

In all cases St Jude mechanical bileaflet prosthetic valve was used. Suturing was done with continuous / interrupted 2-0 ethibond suture in the classical mitral valve replacement patients or after plicating the posterior leaflet with the valve fixation suture in the posterior mitral leaflet area.

All patients were electively ventilated post operatively with inotropic supports being dictated by the hemodynamics of the patient.

The patients out come after surgery where the subvalvar apparatus was preserved either completely or partially were compared against the group in whom the classical technique was followed. The variables assessed were post operative needs for and dosage of multiple inotropic supports, duration of ventilator support.

Post operative left ventricular function was assessed with a pre discharge echo cardiography. The parameters noted were the left ventricular end systolic and end diastolic dimensions post operative left ventricular ejection fraction, reduction in NYHA class.
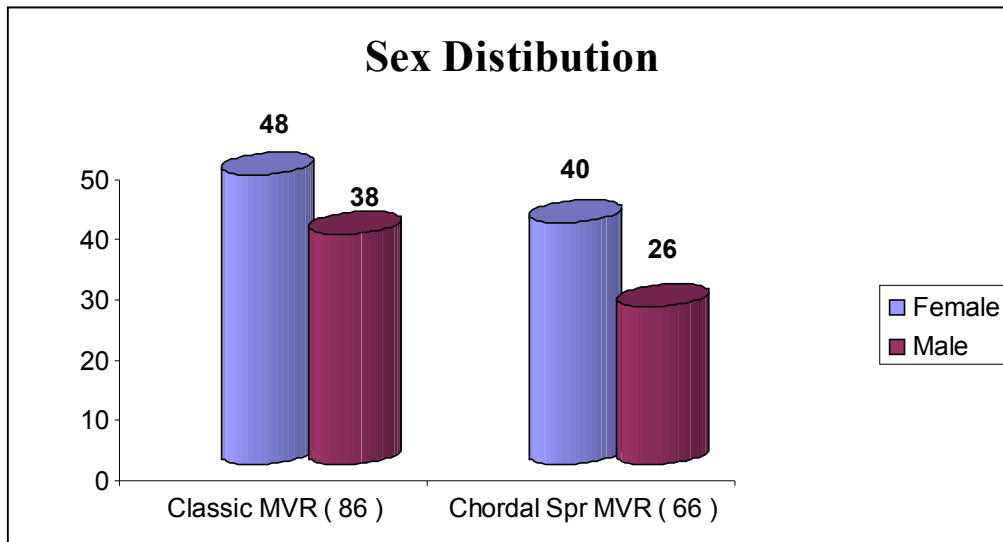
TABLES

TABLE NO 1: AGE DISTRIBUTION

Patient (years)	Classical MVR (86)	Chordal Spr MVR (66)
11 – 20	9	12
21 – 30	27	25
31 – 40	22	14
41 – 50	25	12
51 – 60	3	3
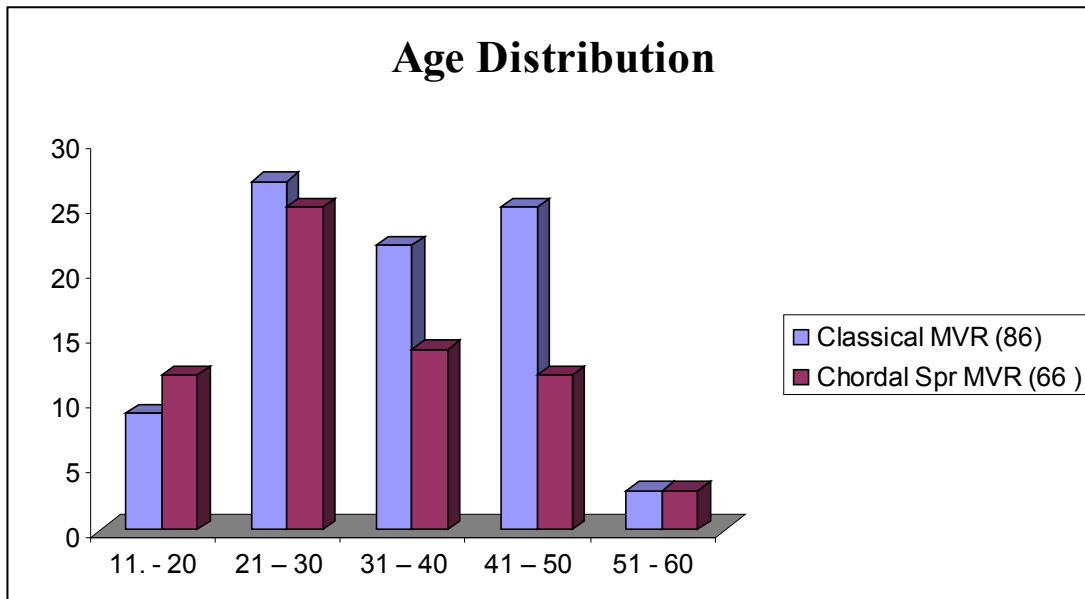
TABLE NO 2: SEX DISTRIBUTION

Sex	Classic MVR (86)	Chordal Spr MVR (66)
Female	48	40
Male	38	26

TABLE NO 3: SYMPTOMS

NYHA Class	Classic MVR (86)	Chordal Spr MVR (66)
I	-	-
II	-	-
III	4	5
IV	82	61
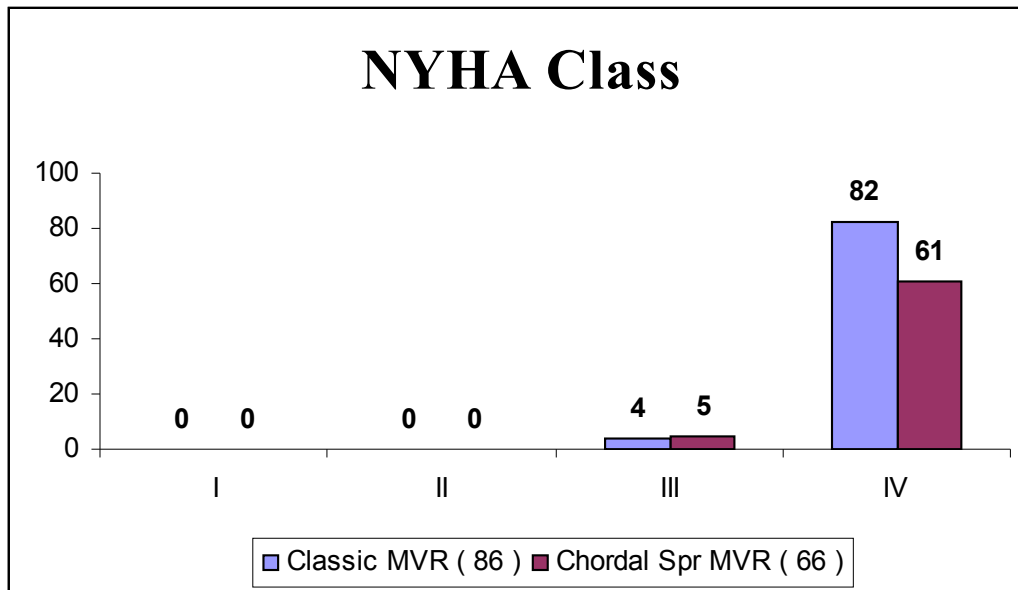
TABLE NO 4: EJECTION FRACTION

Ejection Fraction	Classic MVR (86)	%	Chordal Spr MVR (66)	%
> 60 %	1	1	4	6
50 – 59 %	13	15	24	36
40 – 49 %	63	73	37	56
< 40 %	9	11	1	2

Ejection Fraction chart

- Classic MVR %
- Chordal Spr MVR %
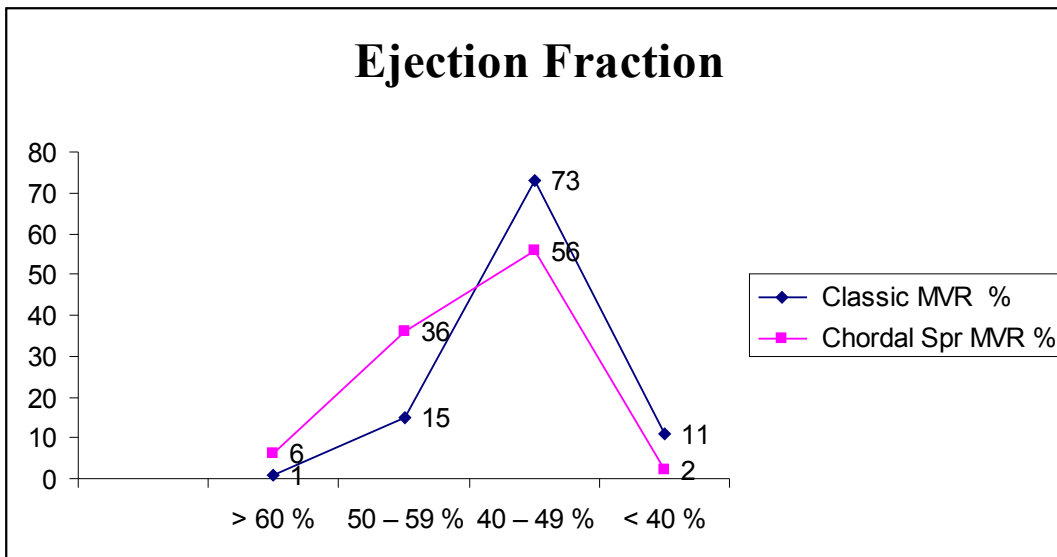
TABLE NO 5: LV END SYSTOLIC DIMENSION

LV eSD (mm)	Classic MVR (86)	%	Chordal Spr MVR (66)	%
> 50	-	-	-	-
40 – 49	11	13	8	12
30 – 39	65	75	55	83
20 – 29	10	12	3	5

LV Systolic Dimension
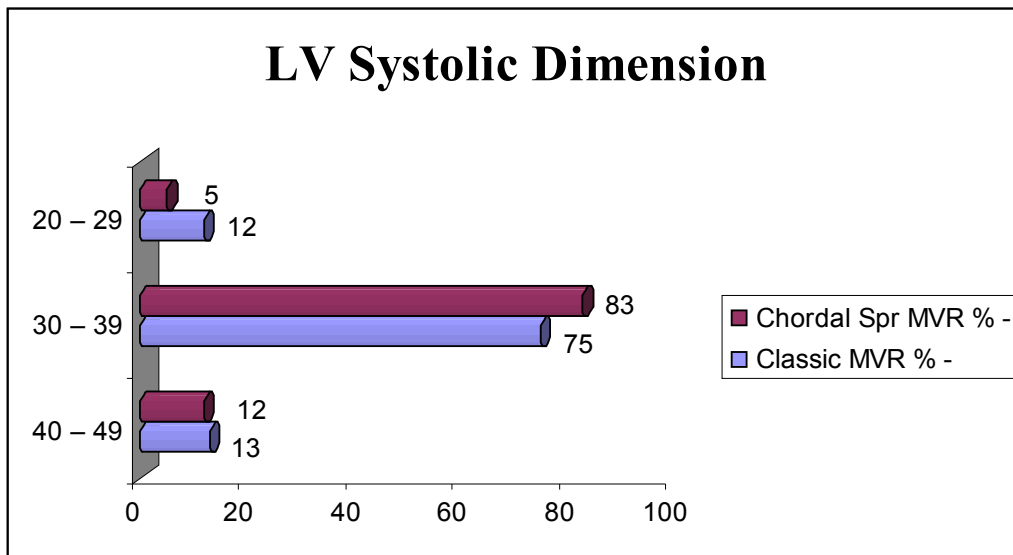
TABLE NO 6: RHYTHM

Rhythm	Classic MVR (86)	%	Chordal Spr MVR (66)	%
Sinus	22	26	22	33
Atrial Fib	64	74	44	67
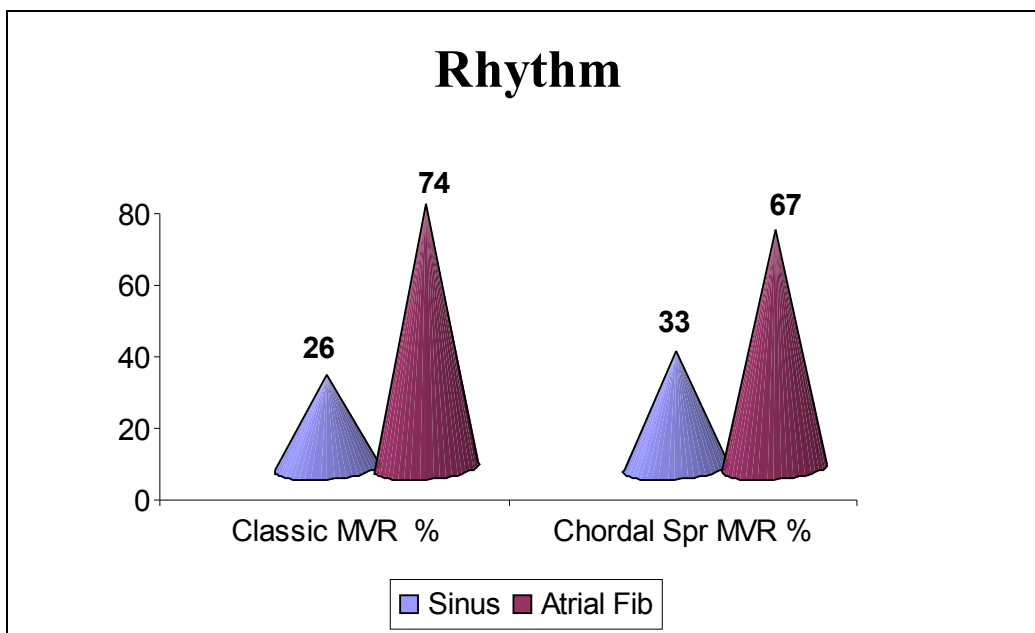
TABLE NO 7: SURGERY

Surgery Technique	No of cases	%
Classic MVR	86	57
Chordal Spr MVR	66	43

Surgery

43%   57%

Classic MVR
Chordal Spr MVR
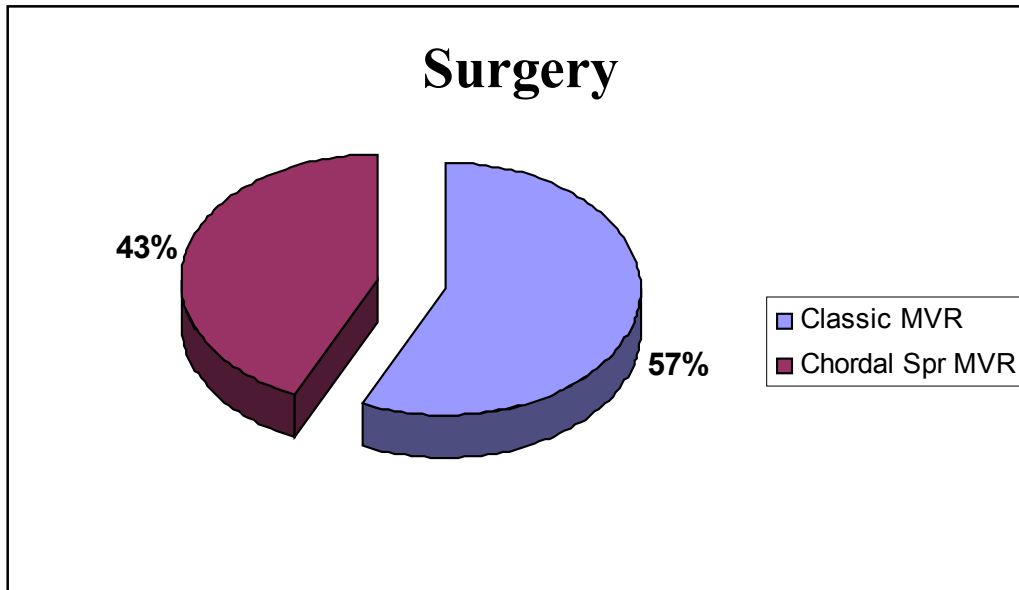
TABLE NO 8: VENTILATOR SUPPORT

Ventilator time	<24 Hrs	%	24 – 48 Hrs	%	>48 Hrs	%
Classic MVR (86)	42	49	43	50	1	1
Chordal Spr MVR (66)	50	76	16	24	-	-

Ventilator Support
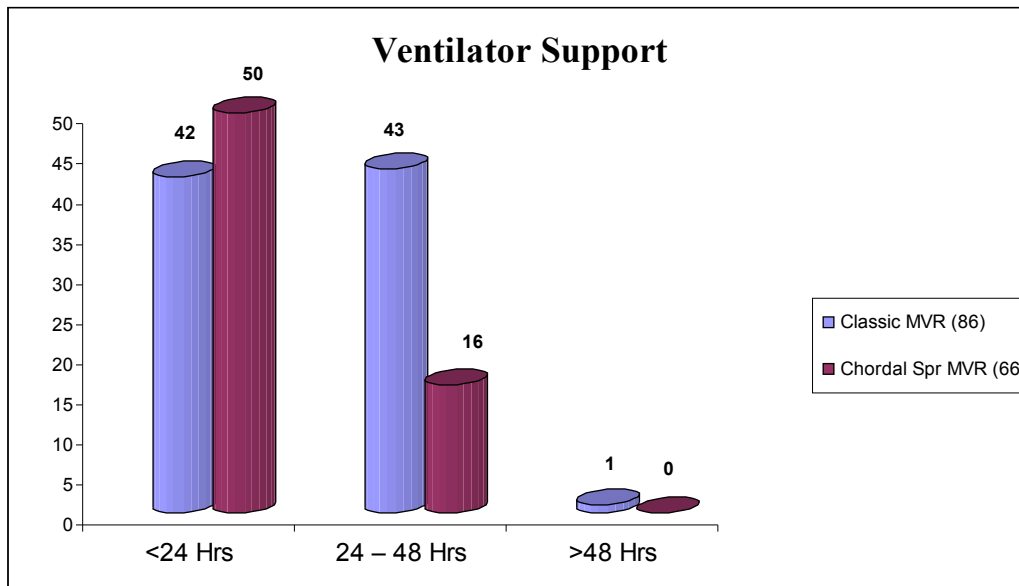
TABLE NO 9: MORBIDITY AND MORTALITY

Morbidity/Mortality	Classic MVR (86)	%	Chordal Spr MVR (66)	%
Re-exploration	2	2	3	5
Wound Infection	5	6	5	8
Low output state	5	6	3	5
Mortality	5	6	2	3
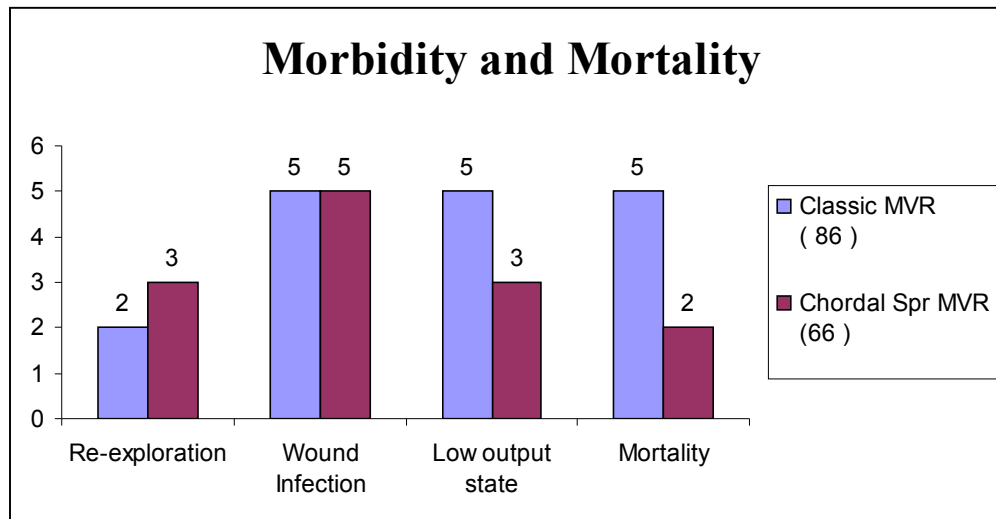
TABLE NO 10: INOTROPIC SUPPORT

Inotrope	Classic MVR (86)	%	Chordal Spr MVR (66)	%
Dop/Dob	49	57	60	91
Dop/Dob+Adr	31	36	5	8
Dop/Dob+Adr+Iso	6	7	1	1
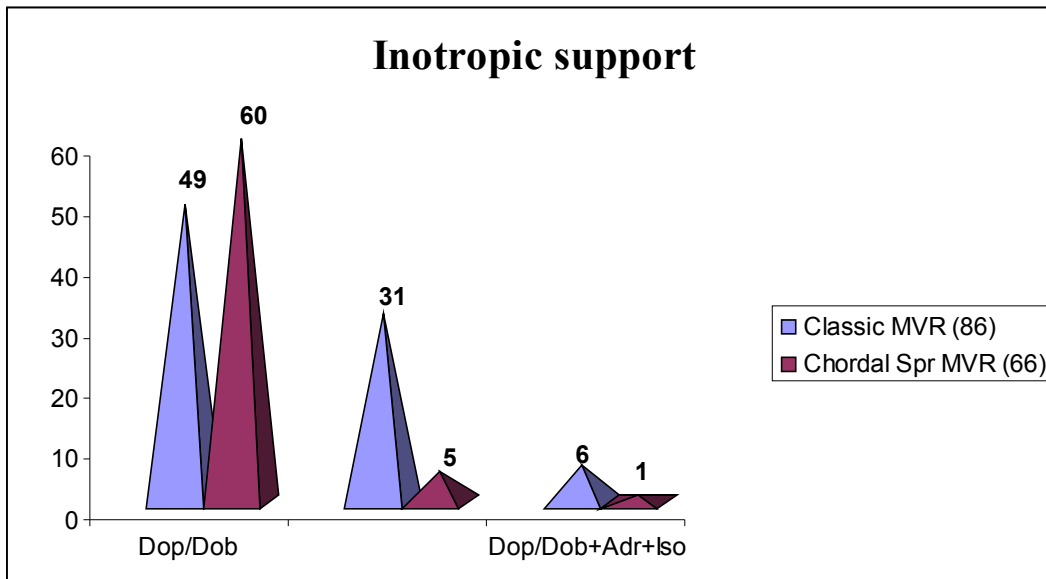
TABLE NO 11: PRE DISCHARGE ECHO

ECHO	EF Decrease	EF Increase/same	LV ESD Increase/same	LV EDD Decrease
Classic MVR (86)	75 (87%)	11 (13%)	33 (38%)	74 (86%)
Chordal Spr MVR (66)	45 (68%)	21 (32%)	7 (11%)	59 (89%)

Pre Discharge ECHO

Bar chart with horizontal axis 0 to 80.

- LV EDD Decrease: Chordal Spr MVR 59, Classic MVR 74
- LV ESD Increase/same: Chordal Spr MVR 7, Classic MVR 33
- EF Increase/same: Chordal Spr MVR 21, Classic MVR 11
- EF Decrease: Chordal Spr MVR 45, Classic MVR 75

Legend: ■ Classic MVR (86) ■ Chordal Spr MVR (66)
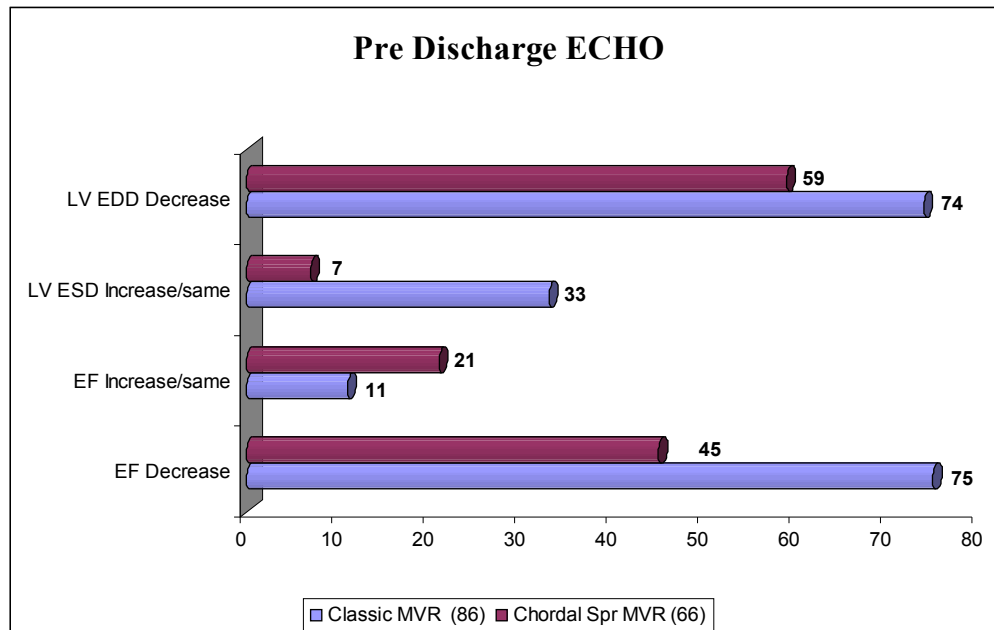
OBSERVATION

A total of 152 patients underwent mitral valve replacement for isolated mitral disease during January 2008 to December 2008. Of this 86 patients (57%) had classical mitral valve replacement and 66 patients (43%) had chordal sparing mitral valve replacement. Lillehei and David technique were followed in chordal preservation group. All patients had St Jude Bileaflet mechanical valve.

TABLE NO 1: AGE DISTRIBUTION.

In both group majority of the patients were in the second decade with a minimum age of 11 yrs and maximum of 59 yrs. Of the 86 patients in classical mitral valve replacement group majority were nearly equally distributed in second, third and fourth decade. Minimum age was 11 yrs and maximum was 59 yrs in this group.
In the chordal sparing group of 66 patients, majority were in the second decade. Minimum age was 13 yrs and maximum was 56 yrs.12 patients were there in the fourth decade and 14 were there in the third decade in chordal sparing group compared to 25 and 22 in the classical group respectively. 3 patients were there in fifth decade in both groups. All patients above 50 yrs had coronary angiogram and had normal coronary arteries.

TABLE NO 2: SEX DISTRIBUTION.

Of the 152 patients 88 were females (58%) and 64 were males (42%). Distribution in the classical mitral valve replacement group was 48 (56 %) females and 38 (44%) males. In the chordal sparing group 40 (61%) were females and 26 (39%) were males.

TABLE NO 3: SYMPTOMS.

Majority of the patients were in NYHA class IV in both group of patients. 82 (95%) and 61 (92%) were in NYHA class IV in classical and chordal sparing group respectively. 4 (5%) and 5 (8%) in classical and chordal sparing mitral valve replacement group respectively presented with NYHA class III symptoms. No patients had class I or class II symptoms.

TABLE NO 4: EJECTION FRACTION.

Majority of the patients had there ejection fraction in 40 to 49% range. In classical group 63 patients (73%) had EF in 40 to 49 % range compared to 37 patients (56%) in chordal sparing group in the same range. In classical group 16 % had EF above 50% and 11% had EF less than 40%. In chordal sparing group 42% were above 50% EF and 2% patients were having EF less than 40%.

TABLE NO 5: LV SYSTOLIC DIMENSION.

Left ventricular end systolic and end diastolic dimensions were measured pre operatively and post operatively. None of the patients had LVeSD above 50 mm. 11 (13%) and 8 (12%) patients had LVeSD in 40 to 49mm range respectively in classical and chordal sparing group. Majority had there LVeSD in 30 to 39mm range which include 65 (75%) in classical group and 55 (83%) in chordal

sparing group. In classical mitral valve replacement group 10 patients had LVeSD in 20 to 29 mm range compared to 3 (5%) in the other group.

TABLE NO 6: RHYTHM.

Atrial fibrillation was present in 64 (74%) of 86 patients in classic mitral valve replacement group and in 44 (67%) of 66 patients in chordal sparing group. In both group 22 patients had sinus rhythm.

TABLE NO 7: TYPE OF SURGERY.

Of the 152 patients with mitral valve replacement, 86 (57%) had classical mitral valve replacement surgery and 66 (43%) had chordal sparing mitral valve replacement surgery.

TABLE NO 8: VENTILATOR SUPPORT.

In the chordal sparing mitral valve replacement group 50(76%) needed less than 12 hrs ventilator support and the remaining 16(24%) needed support more than 24 hrs but less than 48 hrs. In the classical mitral valve replacement group 42(49%) had ventilator support for less than 24 hrs and 44(51%) had support more than 24 hrs out of which one needed support for more than 48 hrs.

TABLE NO 9: MORBIDITY AND MORTALITY.

Five patients were re-explored for excessive drain in chest tube postoperatively of which 2 was in classical and 3 in chordal sparing group respectively. In both group five patients had superficial surgical site infection which responded to medical management. In classical mitral valve replacement group 5 patient had low cardiac output postoperatively as compared to 3 patients in chordal sparing group.
A total of 7 deaths were noted in the total 152 mitral valve replacement group. Of this 5 (6%) were in classic group with mortality rate of 3.28% and 2 (3%) were in chordal sparing group with mortality rate of 1.31%. The deaths were due to low cardiac output syndrome with prolonged ventilator support and multiorgan failure in 5 patients and arrhythmia not amenable to medical management in two patients in classical group.

TABLE NO 10: INOTROPIC SUPPORT

In classical mitral valve replacement group 49(57%) required dopamine / dobutamine or both post operatively compared to 60(91%) in chordal sparing group. 31 (36%) patients required addition of adrenaline in classical group compared to 5 (8%) in chordal sparing group. Of the 86 classic mitral valve replacement 6 (7%) required both adrenaline and isoprenaline and 1 (1%) patient in chordal sparing group required the same.

TABLE NO 11: PRE DISCHARGE ECHO.

There was increase in ejection fraction in 11 (13%) patients in classical group and 21 (32%) patients in chordal sparing group. A reduction in LVeSD was present in 62% of classic group and in 89% in chordal sparing group. The left ventricular end diastolic dimension (LVeDD) decrease was present in 86% of classical group and 89% of chordal sparing group.

DISCUSSION

The majority of the patients in this group were in their second decade of life. This is in contrast to the age group usually observed in the western population where the majority of the population is in the 5th and 6th decade. In the western population the mitral valve prolapse or ischemic mitral regurgitation predominates whereas in Indian population rheumatic heart disease predominates.

Kirklin describes an accelerated form of rheumatic heart disease (RHD) occurs in certain geographical areas where severe mitral stenosis or mitral regurgitation is noticed in a younger age group. The interval before appearance of symptomatic MR is also shorter than for stenosis with a higher chance for previous severe attack of rheumatic fever [40].

The majority of the patients in this group were females. The major etiology of regurgitation or stenosis was rheumatic heart disease. Ling et al in their series of chronic mitral regurgitation noticed 70% of their study group to be males. The majority of cause of regurgitation in their group was a flail leaflet due to ruptured chordae[37]. According to Ikonomidis etal study 60% were females affected by rheumatic etiology and mitral regurgitation being the major presentation[5].

In Demirag etal study, 58.7% were females. The reason for operation was combined mitral stenosis plus regurgitation in 36.96% and etiology being acute rheumatic fever in 75.36%[8].

The majority of patients in this group were in NYHA class IV. This was noticed for both the chordal sparing and classical mitral valve replacement group. No patients were in NYHA class II or class I. This is of particular significance because symptom severity is one of the preoperative predictor of post operative left ventricular function[41].

Grigioni et al in their study of 109 patients with mitral disease noted that the majority of patients in their series were in NYHA class III. Rosen et al in their study of 31 patients with severe mitral regurgitation due to mitral valve prolapse (MVP) were in class III state[42].

The ejection fraction preoperatively in majority of the patient in this study was in the normal or low normal range. Enrique's Sarano et al reported preoperative ejection fraction as a predictor of late out come after mitral valve surgery.

Though ejection fraction is considered to be load dependent it is of considerable significance in post operative predictor of out come following mitral valve replacement. In their work on echo cardio graphic prediction of left ventricular function Zile MR and Fleming et al report an ejection fraction of lesser than 50% as a cut off point in mitral regurgitation wherein left ventricular systolic dysfunction is said to set in.

Zile MR and Fleming et al report larger left ventricular end systolic dimension greater than 50mm or left ventricular end systolic volume index of greater than 50ml/sq m has greater predictor value among all the echo indices for left ventricular dysfunction post mitral valve replacement [43].

Alternative proposed measures include end systolic wall stress index, an end diastolic dimension greater than 70mm, increased left atrial size. Crawford et al in their operative report on 48 patients noted that a low ejection fraction or higher left ventricular end systolic dimension was an important predictor of poor late post operative out come[44].

A total of 108 patients in both the group were in atrial fibrillation with higher no in the classical group. Preoperative atrial arrhythmias have a bearing on the out come in the early phase after surgery[26]. Type of surgery for mitral disease has a bearing on both the early and late out come after mitral valve surgery.

Akins et al indicate that mitral valve replacement is a risk factor for late mortality in comparison with mitral valve repair in univariate analysis. Gilinov et al found by both univariate and multivariate analysis that mitral valve replacement was a risk factor, in comparison to repair[45,46].

Higher inotropic supports were needed in 6 of the chordal preserving mitral valve replacement and 37 of the classical mitral valve replacement patients. They also had to be supported on ventilator for a higher period of time (>24 hrs).

The overall mortality in this series was 4.6% with a mortality of 3.28% in the classical group and 1.31% in the chordal sparing group. The cause of death being low cardiac out put state with multi organ failure. Enriquez Sarano et al in their report from Mayo Clinic report a surgical mortality of 2.6% for valve repair and 10.3% for replacement with a late survival of 58% at 10 years following mitral valve replacement [47].

After valve replacement for chronic mitral regurgitation ejection fraction is lower than preoperatively in most patients. Patients undergoing mitral valve repair have better postoperative ejection indices than similarly matched patients with valve replacement. Left ventricular hypertrophy did not reduce appreciably in mitral valve replacement in comparison to the valve repair group [37, 39].

SUMMARY

One hundred and fifty two cases of mitral valve replacements were carried out between January 2008 and December 2008. Of these eighty six patients had classical mitral valve replacement surgery and sixty six patients had chordal sparing mitral valve replacement. Most of the patients operated were in NYHA class IV. Most of these patients affected were in the second decade with females outnumbering males.

Ejection fraction preoperatively was normal or low normal in majority of patients with both classical mitral valve replacement and chordal sparring mitral valve replacement. One hundred and eight patients had preoperative atrial fibrillation.

In chordal sparing mitral valve replacement group the need for prolonged postoperative ventilator support and higher inotropic supports was not seen. In the classical mitral valve replacement group 44 patients required prolonged ventilation support of greater than 24 hours.

There were seven deaths in this series, two in the chordal sparing mitral valve replacement and five in the classical mitral valve replacement group. Postoperative echo showed a significant improvement in the ejection fraction and decrease in end systolic dimension in the chordal sparing mitral valve replacement group.

CONCLUSION

Mitral valve replacement cannot normalize the life expectancy. Valve replacement has its own limitations. Valve replacement is not the end of the disease but a new beginning of a chronic disease lying dormant, ready to blow off at any time, given a chance.

The best way to address this issue is to stress the importance of mass education, school health education, strict implementation of secondary prophylaxis and an early clinical trial for vaccines for rheumatic fever. In failed cases earlier intervention should be done and not to wait until the heart fails or atrial fibrilation sets in.

Timely intervention when the valve is suitable for conservative procedures and to promote valve sparing surgery as much as possible, there by avoiding the prosthetic valve. In the established cases conservative surgery in the form of chordal preservation should be done with antiarrythymic surgery whenever possible.

In our study of the 66 chordal sparing mitral valve replacement group, better hemodynamics were noted both in the immediate and early post operative period. Chordal sparing mitral valve replacement has been established to protect the left ventricular systolic and diastolic function in the late postoperative period.

A well designed prospective trial with a larger group of patients and a longer follow up period is needed to evaluate further this technique.

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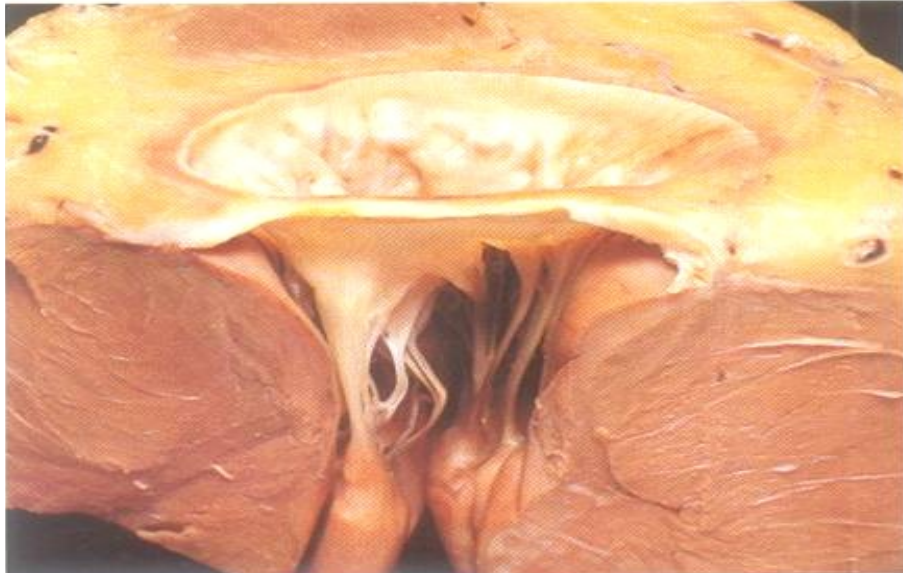
67.   Yun KL, Sintek CF, Miller DC, Pfeffer TA, Kochamba GS, Khonsari S, Zile MR. Randomized trial comparing partial versus complete chordal-sparing mitral valve replacement: effects on left ventricular volume and function J Thorac Cardiovasc Surg 2002; 4 (123): 707-14
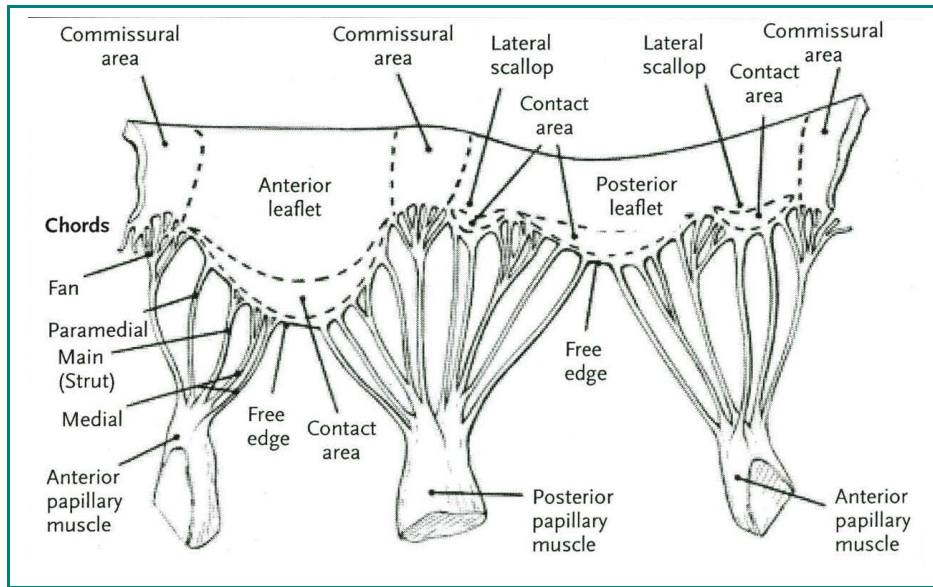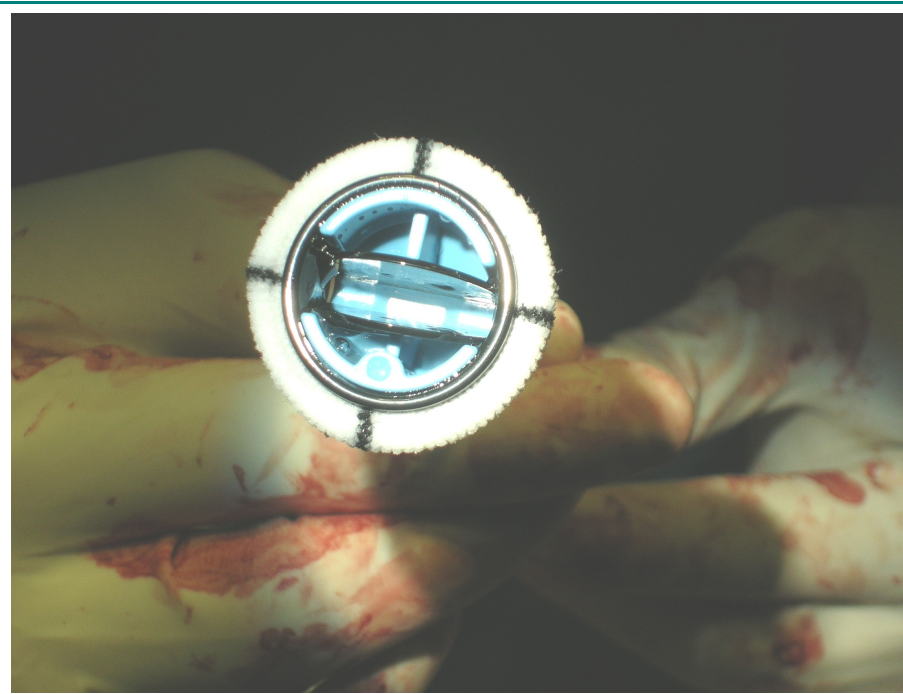
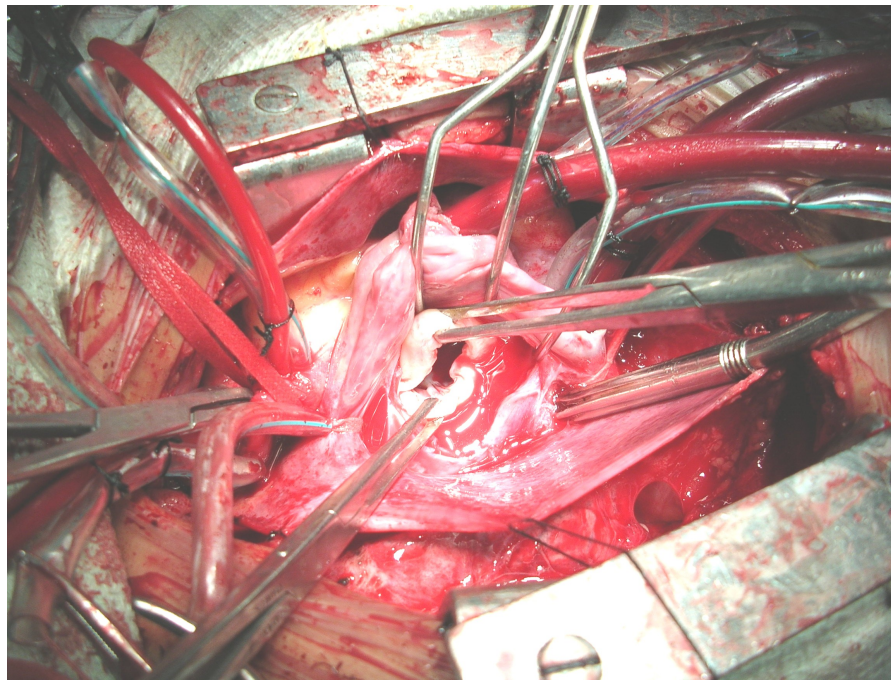**Anatomy of Mitral Valve**
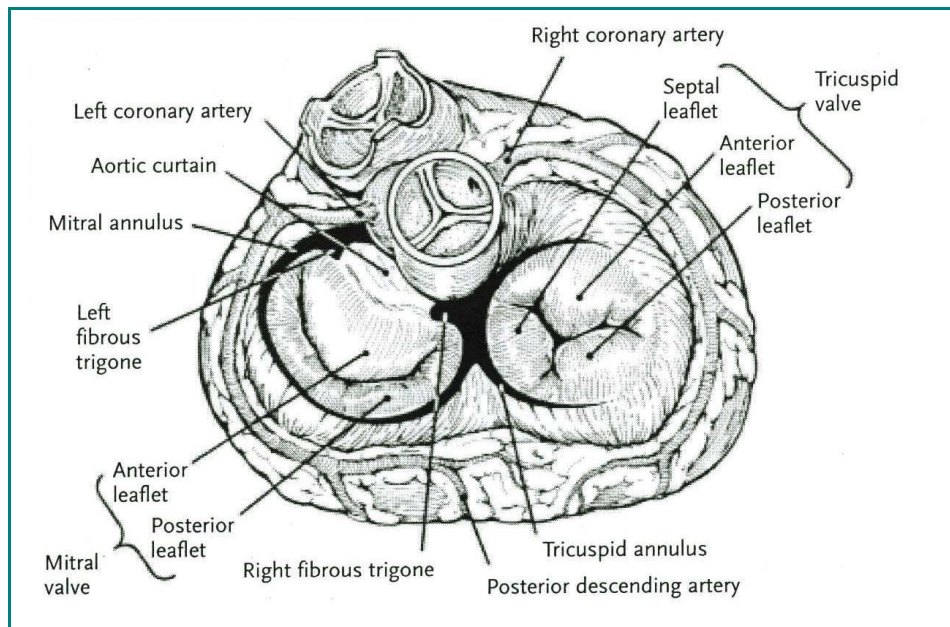
Base of Heart with Atria Removed

**Anatomy of Mitral Valve With
Sub Valvular Apparatus**

View of St.Judes Prosthetic Mitral Valve

Severe Subvalvular Fusion
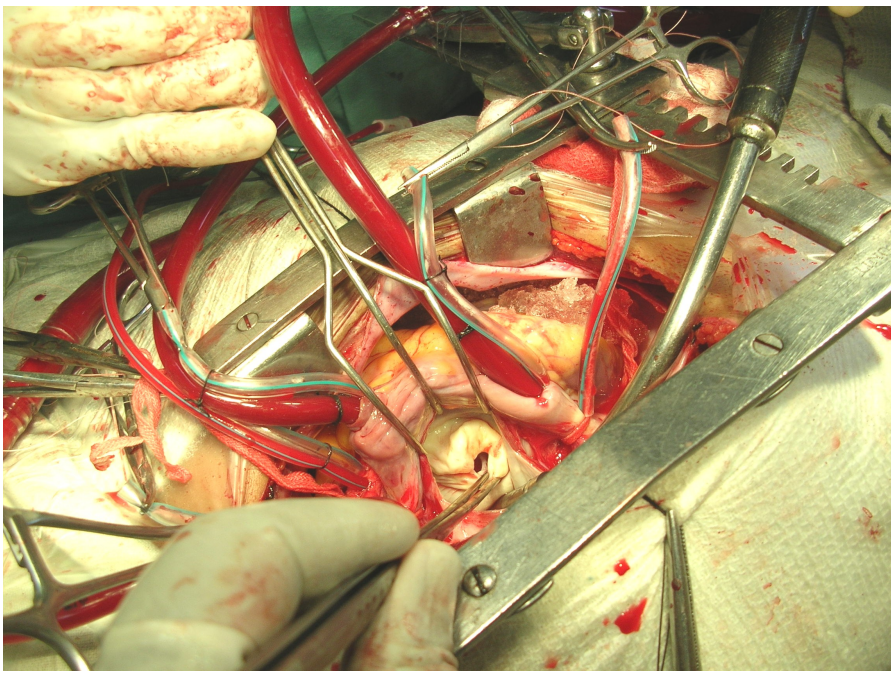
Stenotic Valve
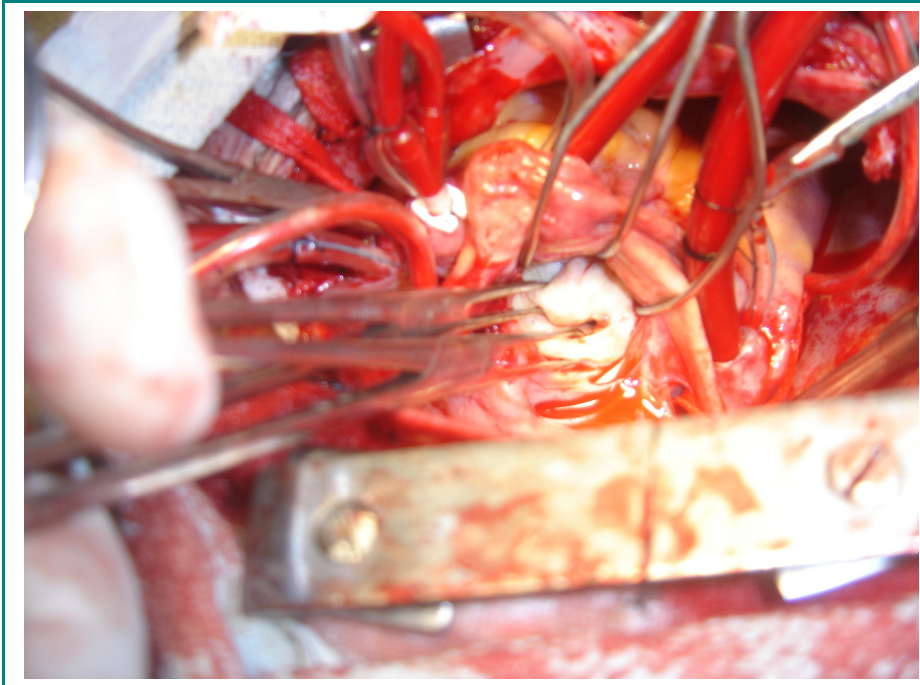
Severe Fibrosis with Subchordal Fusion
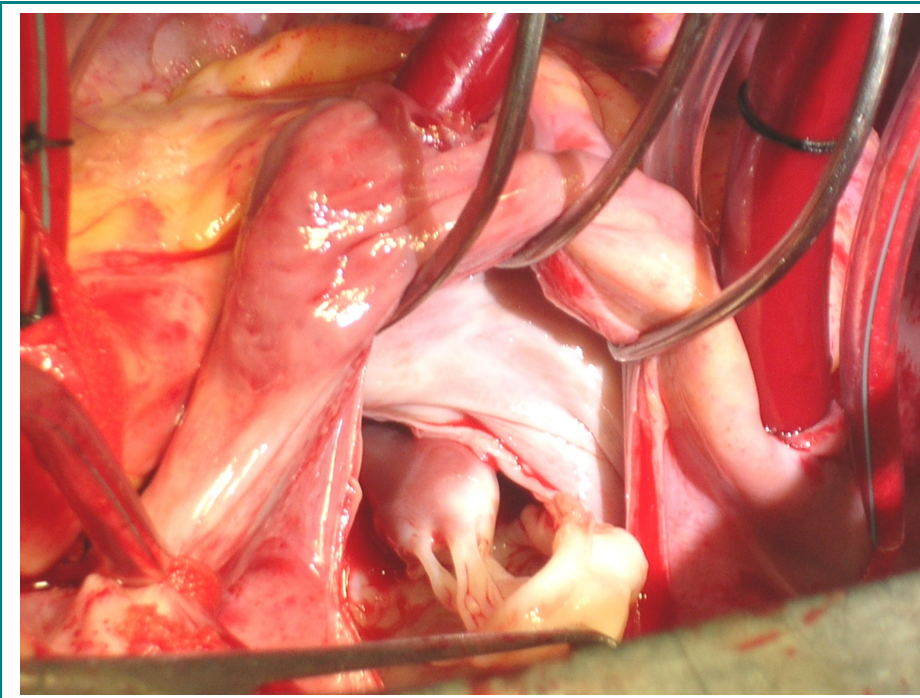
Thickened Valve leaflet with Fused Chordae
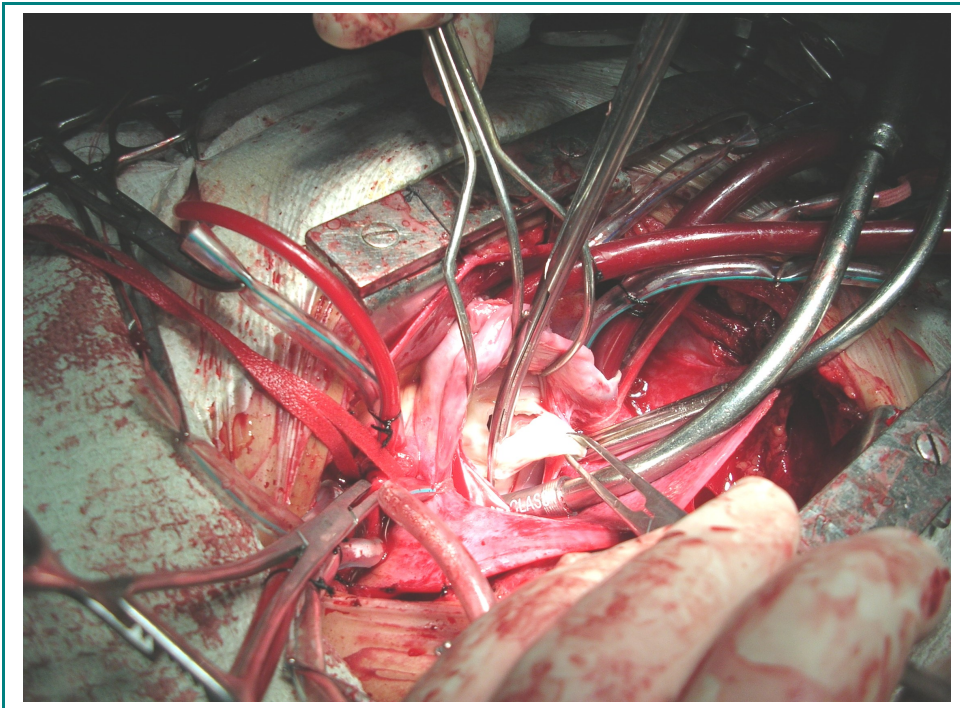
Anterior Mitral Leaflet being Excised
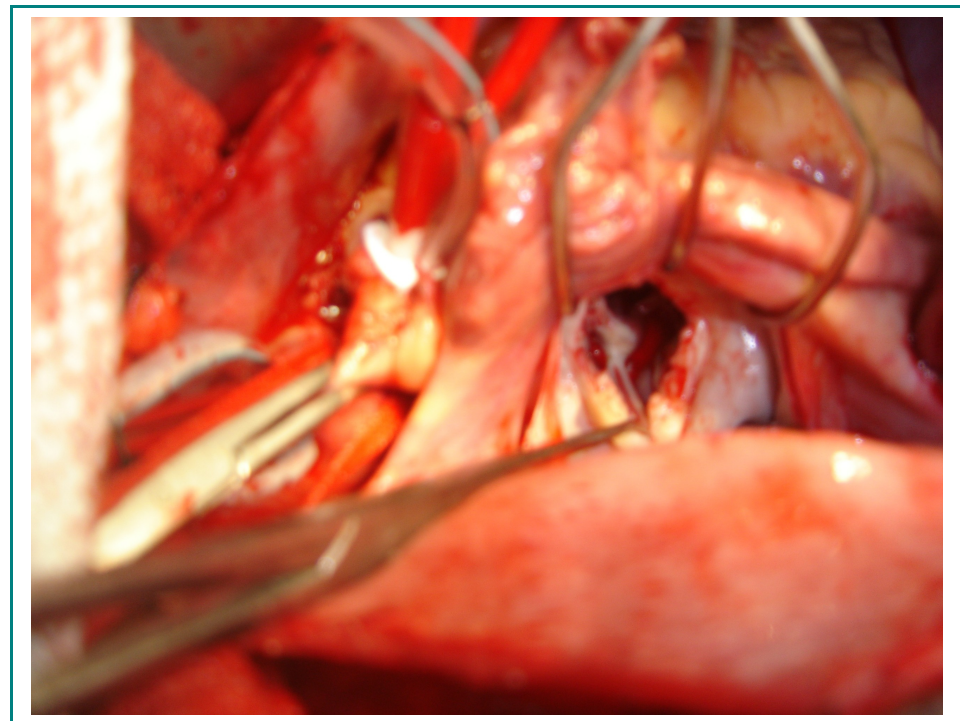
Chordal Preservation after Excision of AML
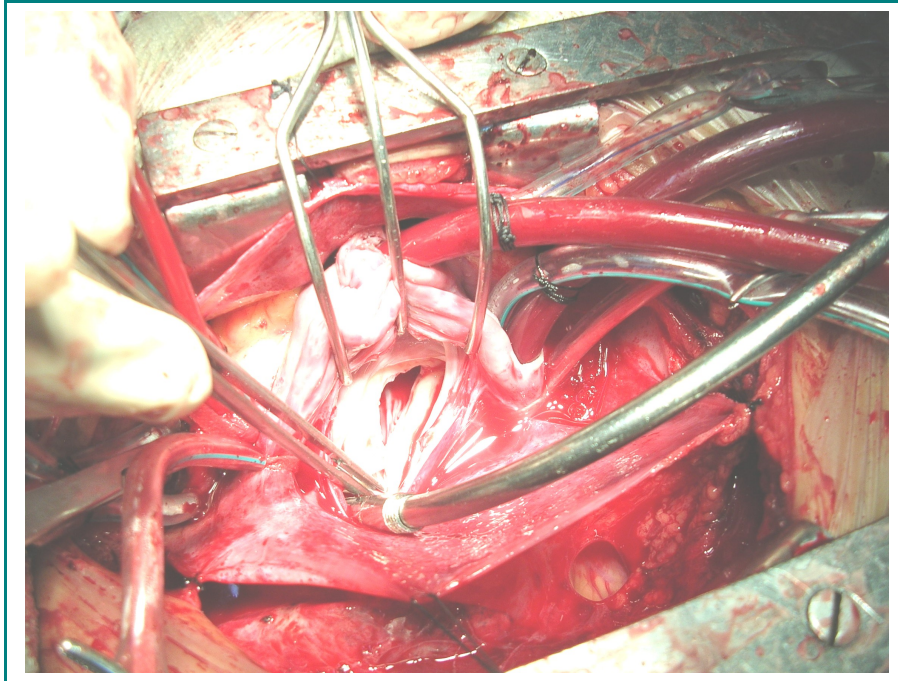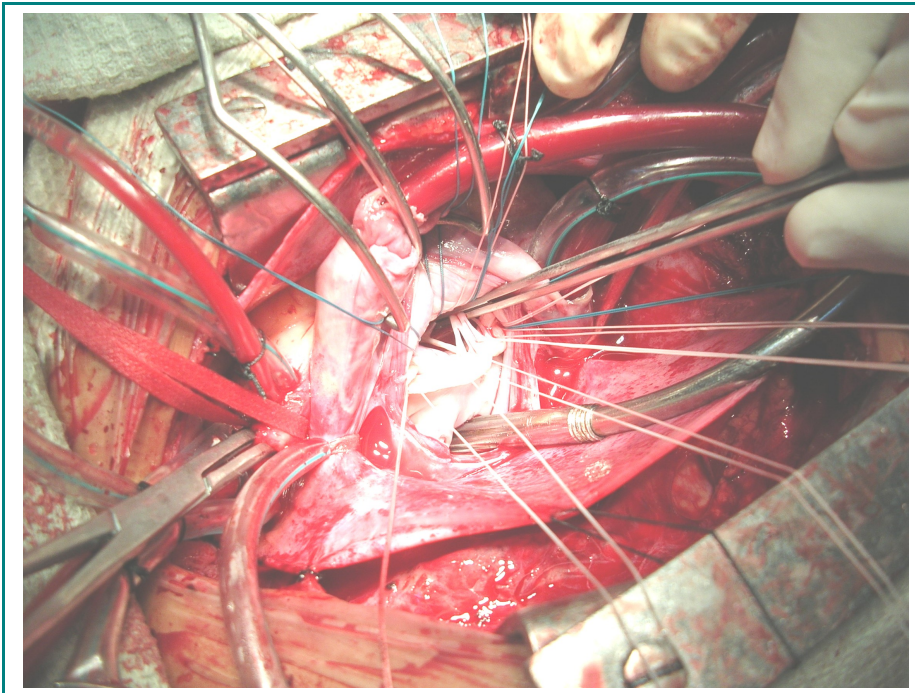
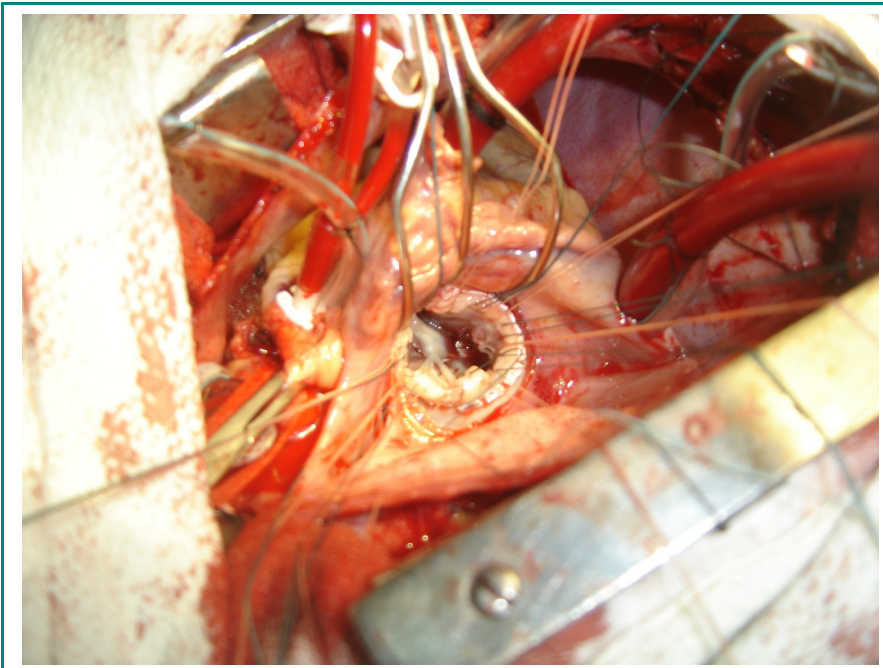**Complete Excision of Subvalvular Apparatus**
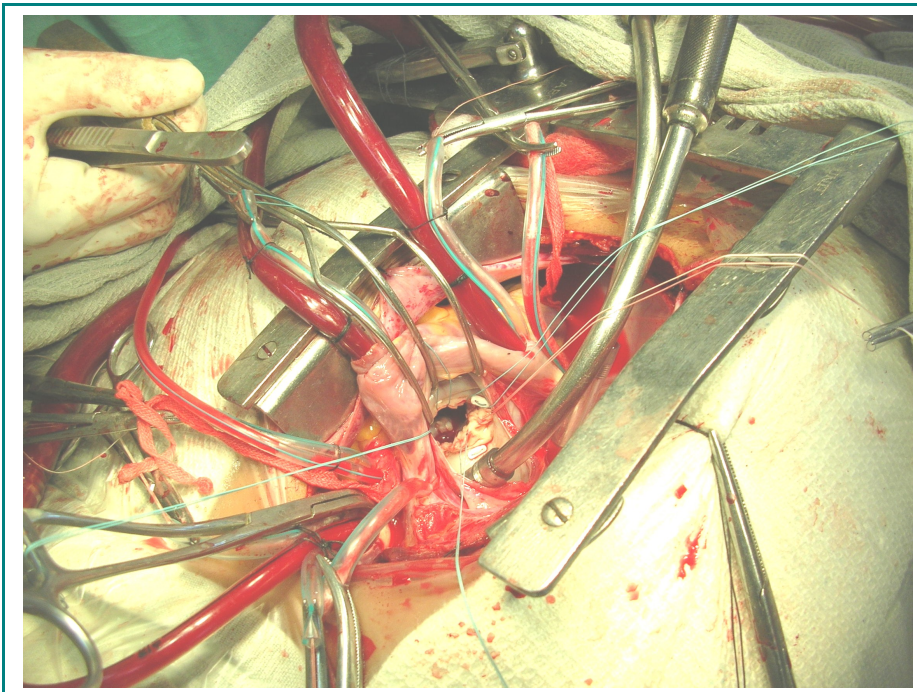
Continuous Suture Technique
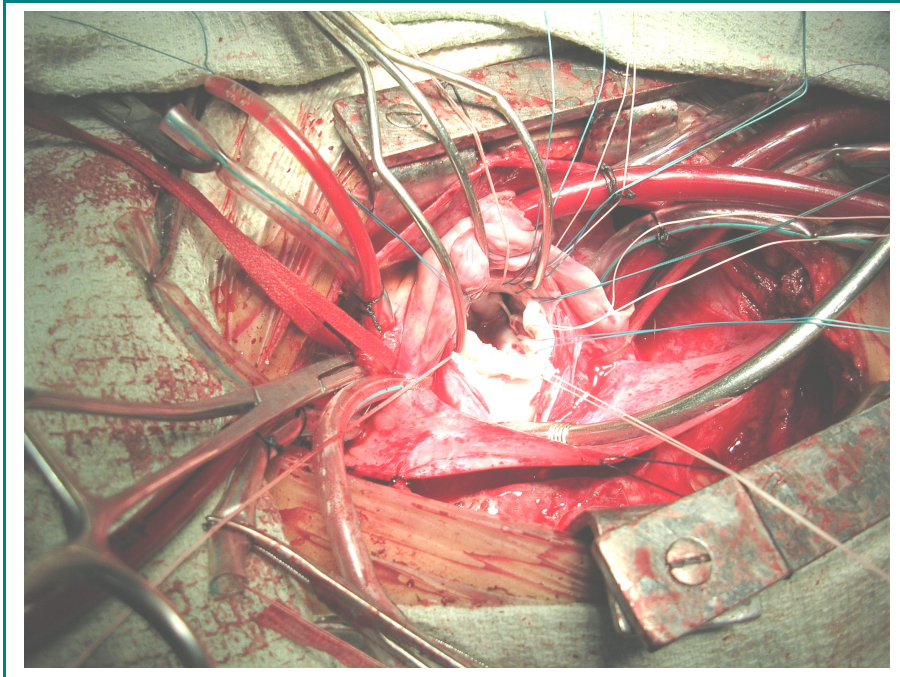
Interrupted Suturing Technique
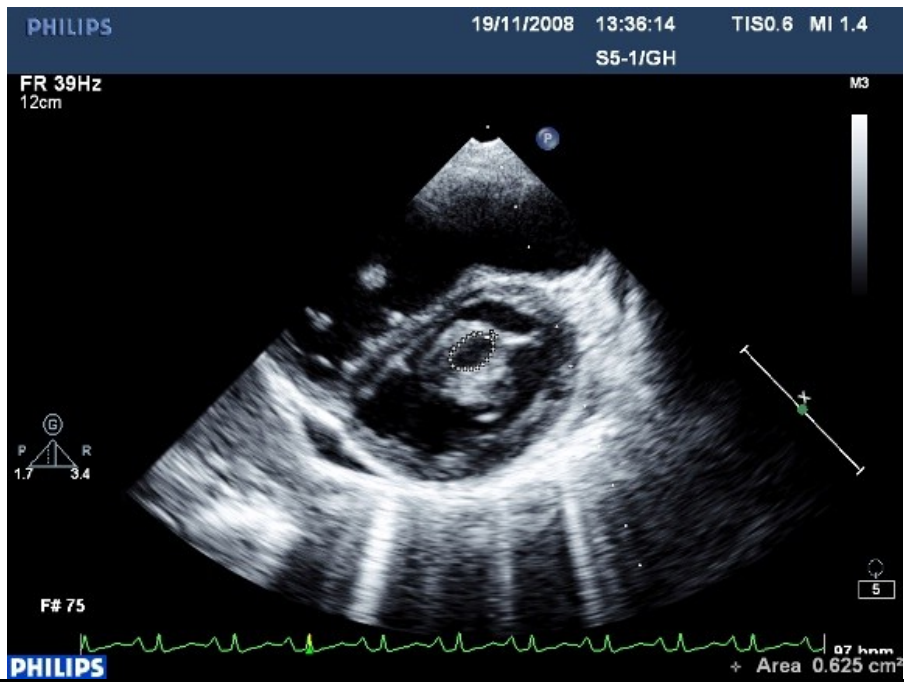
Chordal Preservation Technique (PML Preserved)

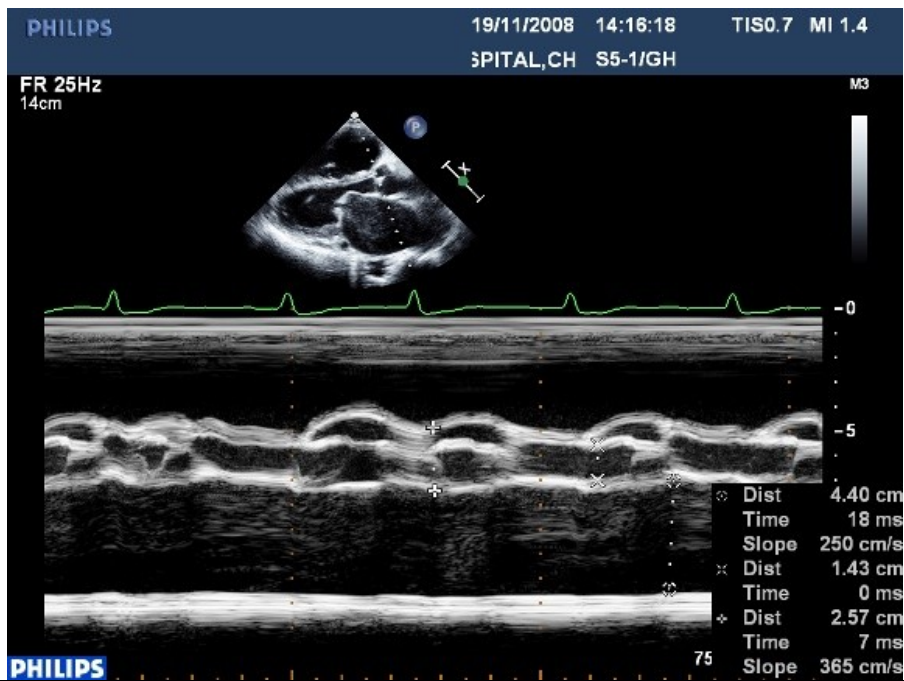**Chordal Preservation Technique with Interrupted Sutures**
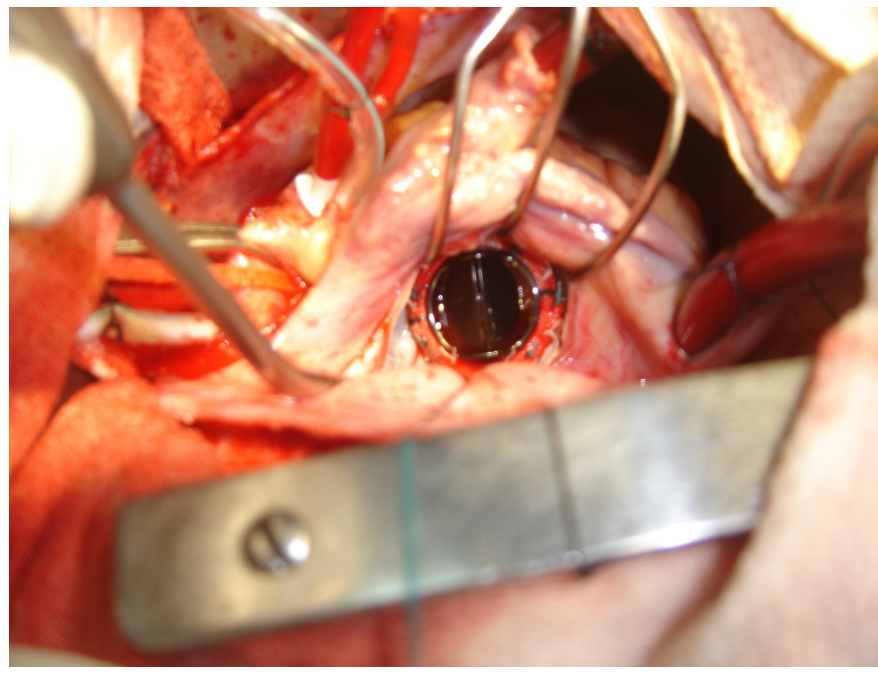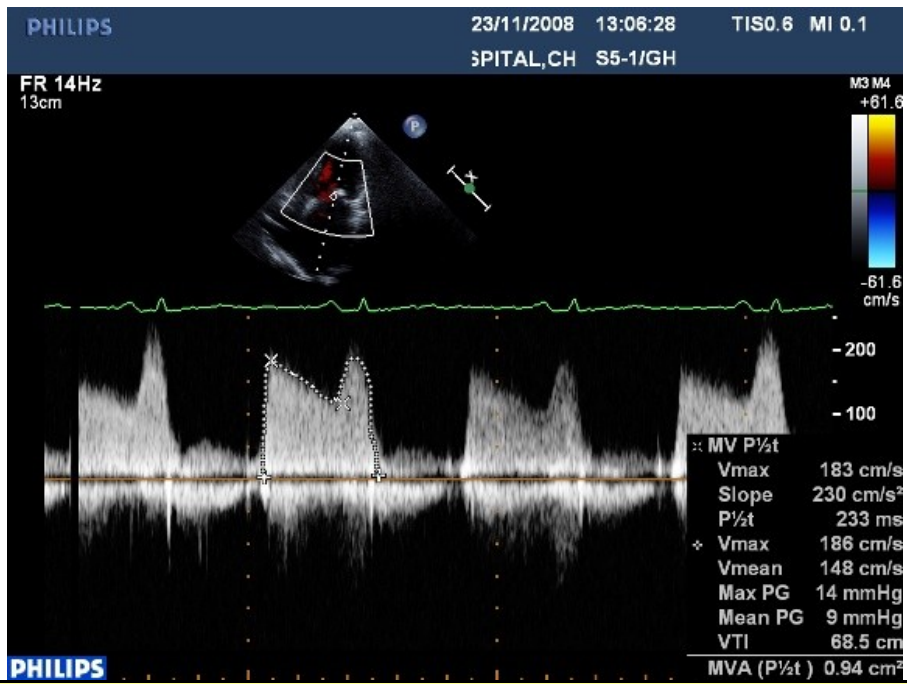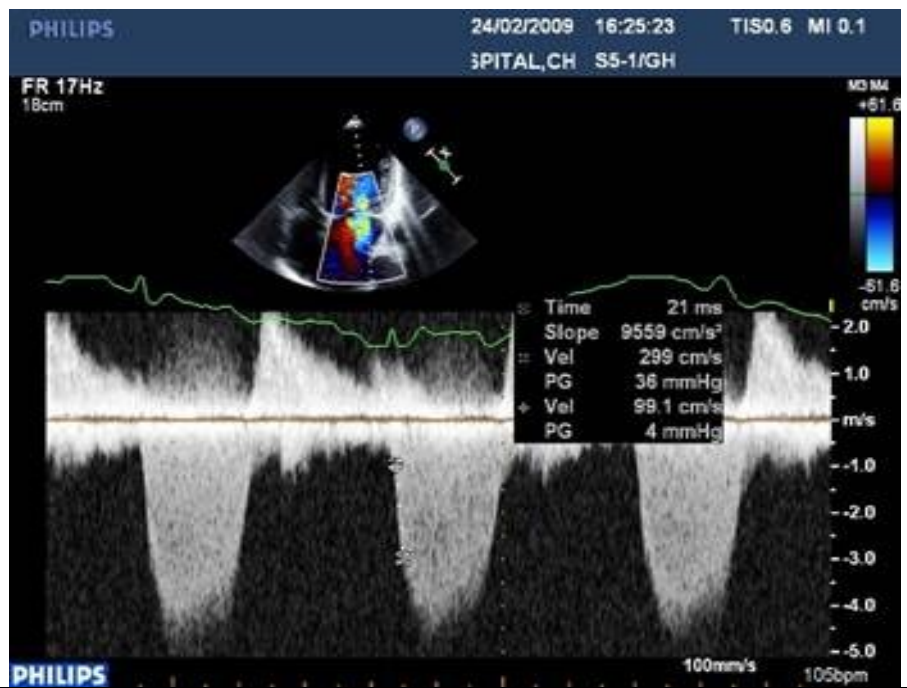
After Mitral Replacement View of St. Jude valve

Trans Thoracic Echo short axis view showing Fish mouth appearance in MS

Trans Thoracic Echo Long Axis view showing Doming of AML

Trans Thoracic Echo Doppler Showing severe MS

Trans Thoracic Echo Doppler Showing Severe MR

APPENDIX - 2

ACC	-	Aortic cross clamp
AHA	-	American Heart Association
AF	-	Atrial fibrilation
AML	-	Anterior mitral leaflet
AV	-	Atrioventricular
EF	-	Ejection fraction
IMR	-	Ischemic mitral regurgitation
LA	-	Left atrium
LV	-	Left ventricle
LVeSD	-	Left ventricular end systolic dimension
LVeDD	-	Left ventricular end diastolic dimension
LVOTO	-	Left ventricular out flow tract obstruction
MRI	-	Magnetic resonance imaging
MR	-	Mitral regurgitation
MS	-	Mitral stenosis
MV	-	Mitral valve
MVP	-	Mitral valve prolapse
MVR	-	Mitral valve replacement
NYHA-		New York Heart Association
PML	-	Posterior mitral leaflet
PTFE	-	Poly tetra fluro ethylene
RHD	-	Rheumatic heart disease
RF	-	Rheumatic fever
SA	-	Sinoatrial