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Three Dimensional Echocardiography a Novel Technique

for Rheumatic Mitral Valve Stenosis Evaluation

Dissertation submitted for DM
(Branch II – Cardiology)

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CERTIFICATE

This is to certify that this dissertation titled "**Three Dimensional Echocardiography a Novel Technique for Rheumatic Mitral Valve Stenosis Evaluation**" submitted by **Dr.S.R.VEERAMANI**, to the faculty of Cardiology, The Tamilnadu **Dr.M.G.R.Medical University**, Chennai in partial fulfillment of the requirement for the award of DM degree Branch II[Cardiology] is a bonafide research work carried out by him under our direct supervision and guidance.

Professor and Head
Department of Cardiology
Madurai Medical College and
Government Rajaji Hospital, Madurai

DECLARATION

I, **Dr.S.R.VEERAMANI**, solemnly declare that the dissertation **titled "Three Dimensional Echocardiography a Novel Technique for Rheumatic Mitral Valve Stenosis Evaluation"** has been prepared by me. This is submitted to The Tamilnadu **Dr.M.G.R.Medical University**, Chennai, in partial fulfillment of the regulations for the award of DM degree Branch II [Cardiology].

Madurai. **Dr.S.R.VEERAMANI**
Date:

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THREE DIMENSIONAL ECHOCARDIOGRAPHY A NOVEL TECHNIQUE FOR RHEUMATIC MITRAL VALVE STENOSIS EVALUATION

INTRODUCTION

Mitral stenosis is characterized by restriction of blood flow from the left atrium (LA) to the left ventricle (LV) as a result of a narrowed mitral passage. It is an acquired valvular defect; it is usually a consequence of rheumatic heart disease, though cases of congenital mitral stenosis are occasionally encountered. The fundamental treatment for rheumatic mitral stenosis[2] is to increase the mitral valve area (MVA) by means of percutaneous transvenous mitral commissurotomy or by surgical valve replacement. In order to establish the time of surgery and an optimal management, it is essential to make an appropriate and accurate assessment of its severity.

At present, the invasive measurement of the mitral valve area is based on the Gorlin formula. This method has been used as the invasive reference method to assess the severity of the rheumatic mitral stenosis. However, it is an invasive method that may result in complications and inaccuracies. Recently, 3DFull volume Planimetry has become an available technique in many echocardiography labs, providing numerous advantages in the assessment of valvular disease.

Zamorano J[14], Cordeiro P, et al studied in 2004,in Eighty patients with Rheumatic Mitral stenosis comprised (76 women; 50.6 +/- 13.9 years). When compared with all other echo-Doppler methods[1], RT3D had the best agreement with the invasively determined MVA (average difference between both methods and limits of agreement: 0.08 cm(2) [-0.48 to 0.6])

In another study, Binder[16]et al. used a first generation real-time 3D echocardiography machine (Volumetrics)in which 3D planimetry proved to be a fast, easy, accurate, and reproducible technique in comparison to 2D planimetry and pressure half-time-derived MVA.

Recent Studies showed that in last few years, three-dimensional echocardiography [2] has become an accurate tool for mitral stenosis assessment. Accuracy of 3DEcho planimetry is superior to the accuracy of the invasive Gorlin's method for mitral valve area (MVA) measurements[16] when a median value obtained from two-dimensional planimetry, pressure half-time[1], and proximal isovelocity surface area method, hence used as the gold standard. 3DEcho improves MVA measurement particularly in less experienced operators compared with experienced operators. 3D Echo also improves the measurement of MVA in patients with calcific mitral stenosis by means of colour planimetry of the flow stream. Comparison of mitral valve volumes measured by 3DEcho in patients with critical and without critical stenosis has shown significantly larger volumes in patients with critical stenosis.

In a recent study, Zamorano et al[14,15]. have studied 29 patients with rheumatic mitral stenosis in a multi-centre study before and after PTMC with second generation trans thoracic real-time 3D echocardiography, found that real-time 3D echocardiography was the most accurate ultrasound technique for measuring MVA, with a better pre- and post-procedural agreement with the invasively (Gorlin) derived MVA, compared to 2D planimetry and pressure half-time-derived MVA[1]. The success rate for real-time 3D echocardiography[15] in 29 consecutive mitral stenosis patients in two centres was 100% for all methods, making real-time 3D echocardiography a feasible technique, with an acceptable acquisition and analysis time of approximately

20 min. Post-PTMC the agreement with the Gorlin-derived MVA was much better, in contrast to 2D planimetry and pressure half-time-derived MVA[1], which may be due to the hemodynamic and compliance changes affecting the latter, as was observed before.

In our part of the world there were limited number of studies even though there is much necessity to know more in detail about the Rheumatic Mitral valve disease[15] which is more prevalent.

REVIEW OF LITERATURE

ANATOMY OF THE MITRAL VALVE

It is important to recognize that the leaflets of the mitral valve constitute only a portion of the mitral valve apparatus and that diseases resulting in mitral dysfunction often are caused by abnormalities in the overall apparatus rather than in the actual leaflets. The components of the mitral apparatus include the mitral annulus, the leaflets, chordate tendineae , papillary muscles, and the underlying ventricular wall. Pathologic changes in any of these components of the mitral valve apparatus can result in mitral valve dysfunction. The classic form of mitral valve disease is rheumatic heart disease, which involves predominantly the leaflets and chordae.

There are two mitral valve leaflets, typically referred to as anterior and posterior. The mitral leaflet should be viewed not as a two-leaflet structure but as a six-scallop structure. Clinically, the most easily understood and clinically useful description of mitral valve anatomy involves dividing it into six scallops, three each for the anterior and posterior leaflet, designated as scallop 1, 2, and 3. Scallop 1 is most lateral and scallop 3 is most medial. Chordae attach throughout the entire length of the coaptation line of each of the mitral valve leaflets and insert into the tips of the papillary muscles.

Anatomically, there are two major papillary muscles, each of which may have several heads. The antero lateral papillary muscle provides chordae to the antero lateral half of both mitral leaflets. The postero medial papillary muscle provides chordae to the postero medial aspect of both leaflets. There is substantial variability from patient to patient in the exact number of chordae and the percentage of chords

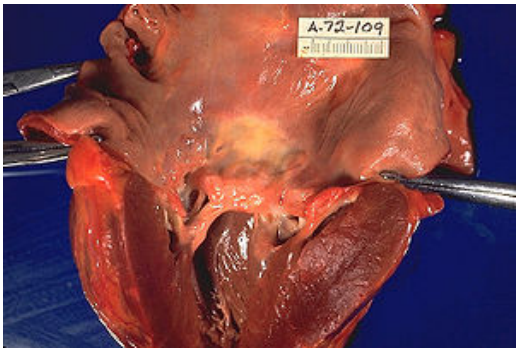
that are devoted to the anterior and posterior leaflets, but in general both papillary muscles provide chordal attachments to a portion of each of the leaflets.

Approximately 60% of patients with pure mitral stenosis have a history of rheumatic fever[2], although in some parts of the developing world, the initial episode of rheumatic fever may be unrecognized due to scarcity of health-care resources. The diagnosis of rheumatic fever is made using the **Jones criteria** in a patient with a history of streptococcal infection. To make the diagnosis, two major criteria or one major and two minor criteria must be met.

Jones Criteria for Diagnosis of Rheumatic Fever. To Make the Diagnosis of Rheumatic Fever, Either Two Major or One Major and Two Minor Criteria Must be Present

Major criteria	Minor criteria
Carditis	Arthralgias
Polyarthritis	Fever
Chorea	Elevated C-reactive protein
Erythema marginatum	Elevated erythrocyte sedimentation rate
Subcutaneous nodules	Prolonged PR interval

Figure.1 Macroscopic appearance of Rheumatic Mitral Valve[2]



In addition to the major and minor criteria listed above, there must be evidence of streptococcal infection 2–4 weeks earlier (either a positive throat culture for group A beta-hemolytic streptococci, a positive rapid streptococcal antigen test, or elevated streptococcal antibody titers). In developing areas of the Indian subcontinent, Middle East, and Asia, rheumatic fever remains more frequent.

Etiology of Mitral Stenosis[2]

Acquired

- Rheumatic heart disease (vast majority of cases)
- Calcific mitral stenosis
- Hypereosinophilia
- Cafergot toxicity
- SLE, malignant sarcoid, mucopolysaccharidosis
- Active infective endocarditis, Whipple's disease

Congenital

- Parachute mitral valve complex
- Shone's complex: parachute mitral valve with supra-valvar mitral ring, sub-valvular aortic stenosis and coarctation of the aorta

In Rheumatic Mitral Stenosis, at autopsy with characteristic findings like thickened mitral valve, thickened chordae tendineae, hypertrophied left ventricular myocardium are seen. Almost all cases of mitral stenosis are due to disease in the heart secondary to rheumatic fever and the consequent rheumatic heart disease. Uncommon causes of mitral stenosis are calcification of the mitral valve leaflets, and as a form of congenital heart disease. However, there are primary causes of mitral stenosis that emanate from a cleft mitral valve. Other causes include Bacterial endo carditis where the vegetations may favor increased risk of stenosis. It is the most common valvular heart disease in pregnancy.

Patho physiology

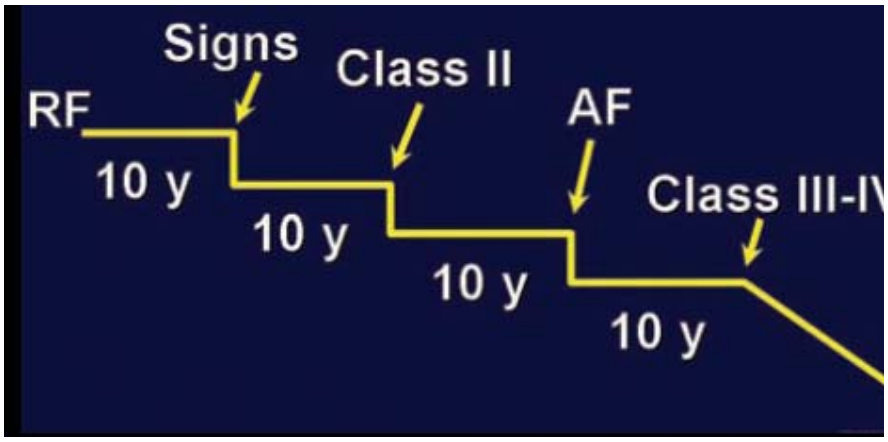
The normal area of the mitral valve orifice is about 4 to 6 cm^2. In normal cardiac physiology, the mitral valve opens during left ventricular diastole, to allow

blood to flow from the left atrium to the left ventricle. A normal mitral valve will not impede the flow of blood from the left atrium to the left ventricle during (ventricular) diastole, and the pressures in the left atrium and the left ventricle during ventricular diastole will be equal. The result is that the left ventricle gets filled with blood during early ventricular diastole, with only a small portion of extra blood contributed by contraction of the left atrium (the "atrial kick") during late ventricular diastole. When the mitral valve area goes below 2 cm^2, the valve causes an impediment to the flow of blood into the left ventricle, creating a pressure gradient across the mitral valve. This gradient may be increased by increases in the heart rate or cardiac output. As the gradient across the mitral valve increases, the amount of time necessary to fill the left ventricle with blood increases. Eventually, the left ventricle requires the atrial kick to fill with blood. As the heart rate increases, the amount of time that the ventricle is in diastole and can fill up with blood (called the diastolic filling period) decreases. When the heart rate goes above a certain point, the diastolic filling period is insufficient to fill the ventricle with blood and pressure builds up in the left atrium, leading to pulmonary congestion.

When the mitral valve area goes less than 1 cm^2, there will be an increase in the left atrial pressures (required to push blood through the stenotic valve). Since the normal left ventricular diastolic pressures is about 5 mmHg, a pressure gradient across the mitral valve of 20 mmHg due to severe mitral stenosis will cause a left atrial pressure of about 25 mmHg. This left atrial pressure is transmitted to the pulmonary vasculature and causes pulmonary hypertension. Pulmonary capillary pressures in this level cause an imbalance between the hydrostatic pressure and the oncotic pressure, leading to extravasations of fluid from the vascular tree and pooling of fluid in the lungs (congestive heart failure causing pulmonary oedema). The constant pressure

overload of the left atrium will cause the left atrium to increase in size. As the left atrium increases in size, it becomes more prone to develop atrial fibrillation. When atrial fibrillation develops, the atrial kick is lost (since it is due to the normal atrial contraction). In individuals with severe mitral stenosis, the left ventricular filling is dependent on the atrial kick. The loss of the atrial kick due to atrial fibrillation can cause a precipitous decrease in cardiac output and sudden congestive heart failure. Patients with mitral stenosis prompts a series of hemodynamic changes that frequently cause deterioration of the patient's clinical status. A reduction in cardiac output, associated with acceleration of heart rate and shortening of the diastolic time, frequently leads to congestive heart failure. In addition, when AF sets in, systemic embolization becomes a real danger. Mitral stenosis typically progresses slowly (over decades) from the initial signs of mitral stenosis to NYHA functional class II symptoms to the development of atrial fibrillation to the development of NYHA functional class III or IV symptoms. Once an individual develops NYHA class III or IV symptoms, the progression of the disease accelerates and the patient's condition deteriorates.

Natural History of Mitral Stenosis



Secondary Features of Mitral Stenosis

Chronic mitral stenosis results in several common and easily recognized secondary features, the overwhelming majority of which are related to an increase in left atrial pressure. Chronic elevation in left atrial pressure results in left atrial dilation and eventual fibrosis of the atrial myocardium. Over time, fibrosis of the atrial myocardium results in decreased atrial contraction and serves as a substrate for development of atrial fibrillation. Dilation of the left atrium occurs both in the atrial body and left atrial appendage. The combination of atrial and atrial appendage dilation with decreasing mechanical function results in stasis of the blood with an enhanced propensity to thrombus formation, most commonly in the left atrial appendage. This typically appears as a swirling mass of echoes in the body of the left atrium, referred to as spontaneous echo contrast, and is often maximal in the left atrial appendage.

Current opinion suggests that spontaneous echo contrast and stasis of the blood are precursors to thrombus formation in the left atrium that carry a nearly equivalent risk of thrombo embolic disease, especially if seen in the presence of atrial fibrillation. When evaluating a patient for a possible left atrial appendage thrombus, it is important to recognize the range in anatomic variability of the atrial appendage. Traditionally, the left atrial appendage has been considered a single-lobe structure with varying degrees of trabeculation due to pectinate muscles. It is now well recognized that the left atrial appendage has multiple lobes in a substantial percentage (>30%) of patients. This raises several concerns when evaluating patients for a left atrial appendage thrombus. The first is that all lobes of the appendage must be identified and examined. The second issue is the need to recognize the septation tissue between appendage lobes as normal tissue and not as protruding thrombus.

Atrial Fibrillation

A frequent sequel of left atrial dilation is atrial fibrillation, which can be either intermittent or persistent. In the presence of atrial fibrillation, there is a loss of organized mechanical activity of the left atrium. This intensifies the tendency to form spontaneous echo contrast and thrombus. The fibrillatory mechanical activity of the atrium can be appreciated by either two-dimensional visualization or M-mode echocardiography of the left atrial wall. Additionally, Doppler echocardiography at the mouth of the atrial appendage reveals indirect evidence of the reduction in mechanical force due to atrial fibrillation. Marked reduction in velocity and volume of flow out of the left atrial appendage compared with velocities seen in normal sinus rhythm and is the anatomic/physiologic basis for stasis and formation of clot. Patients with atrial fibrillation and relatively intact atrial appendage transport function as documented by preserved emptying velocities (>50 cm/sec) are less likely to have spontaneous contrast (and presumably thrombosis) than are those with reduced atrial appendage velocities.

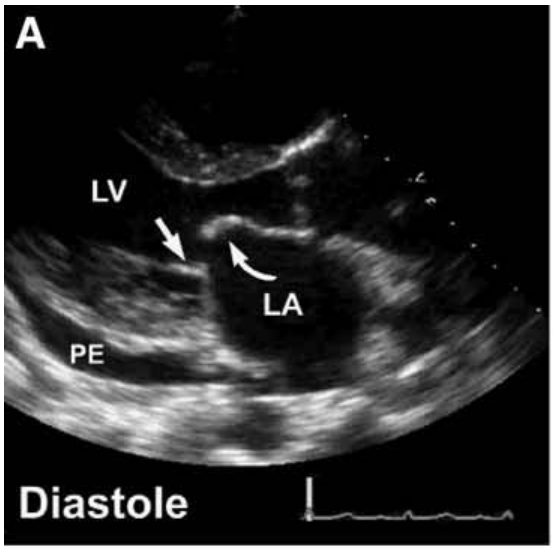
Secondary Pulmonary Hypertension

An additional sequel of long-standing severe mitral stenosis is secondary pulmonary hypertension. In the early phases, this is due in large part to reactive changes in pulmonary vascular resistance and is reversible with correction of mitral stenosis. In long-standing severe mitral stenosis, a fixed component occurs, and in this instance, pulmonary artery systolic hypertension may be only partially reversible. Echo cardio graphic manifestations of secondary pulmonary hypertension in mitral stenosis are similar to those seen in pulmonary hypertension of any cause. Concurrent tricuspid regurgitation is present in the majority of these patients, usually due to right ventricular dilation and less often due to direct involvement of the tricuspid valve by the rheumatic process.

Two-Dimensional Echocardiography in Rheumatic Mitral Stenosis[10,21]

The classic findings of rheumatic mitral stenosis involve thickening and fusion of the mitral valve commissural edges and chordae. This results in characteristic abnormalities of the mitral leaflet opening motion. Normally, the anterior and posterior leaflets open with a motion pattern that involves maximal excursion at the leaflet tips. In mitral stenosis, due to commissural fusion, the leaflets open with a doming motion. In rheumatic heart disease, the open anterior leaflet has also been described as having a hockey stick appearance. Initially, this results in reduction of the orifice and conversion of the mitral leaflet chordal apparatus from a tubular channel to a funnel-shaped orifice. It should be recognized that the limiting factor in flow from the left atrium to the left ventricle will be the orifice of the mitral valve and chordae at their junction. The degree of chordal thickening and mitral valve commissural fusion is highly variable. Over time, there is progressive fibrosis at the initial site of fusion as well as throughout the more distal chordae and more proximal leaflets. Eventually, this results in stiffening and calcification of these structures and were recorded in patients with varying degrees of rheumatic mitral valve involvement. The relatively pliable belly of the mitral valve leaflets with the disease process limited to the tips and chordae. Later stages results in which there is substantial fibrosis or calcification.

PLAXView(A)Diastole[3],(B)Systole[3]



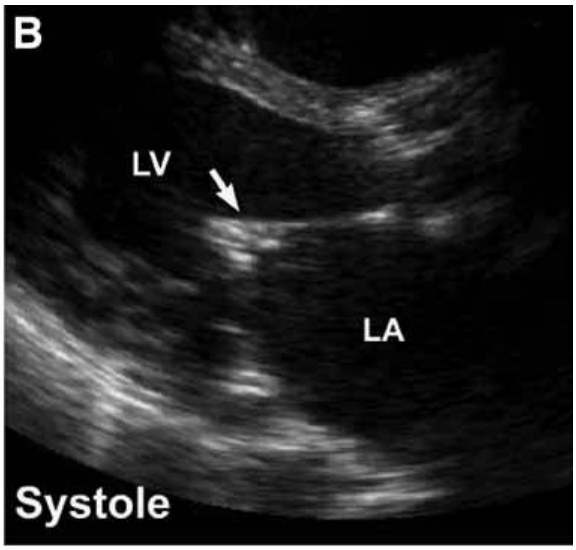


Figure. 2 **Figure. 3**

Transthoracic parasternal long-axis view echocardiogram recorded in a patient with rheumatic heart disease and mitral stenosis. In this image, recorded in early diastole, note the doming motion of the anterior mitral valve leaflet with restriction of motion at the tips. The belly of the leaflet (arrows) is pliable, and there is little or no fibrosis, calcification, or thickening of the leaflets. Also note the secondary dilation of the LA.

PSAX View

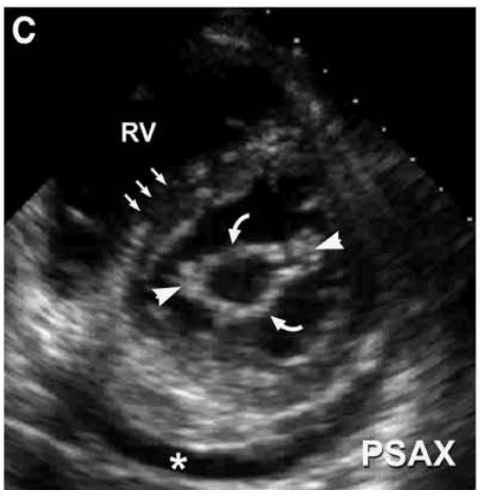


Figure .4

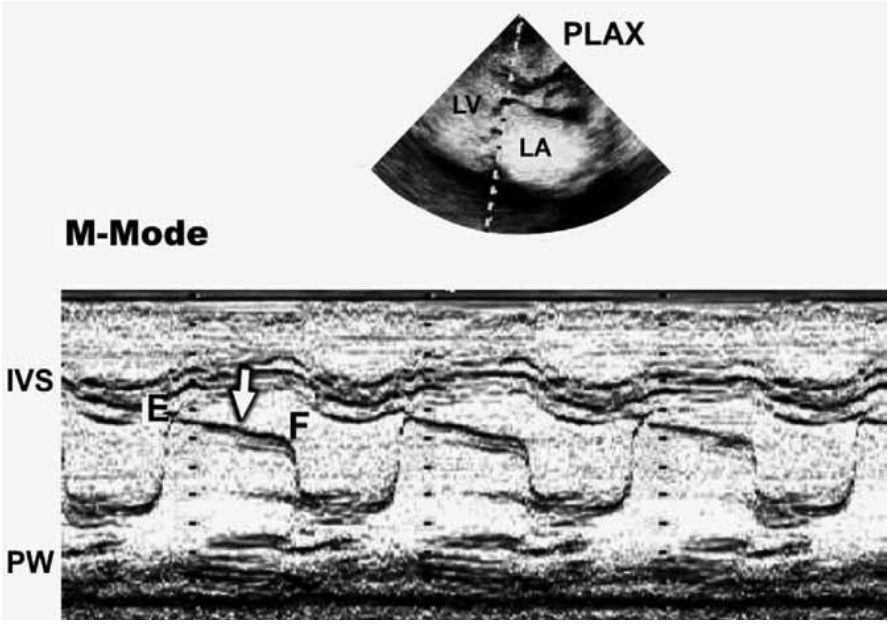


Figure .5

M-mode and 2D echo features of Rh.MS include the following

1. Thickened and calcified mitral leaflets and subvalvular apparatus
2. Decreased E-F slope (M-mode)
3. Hockey-stick appearance of the anterior mitral leaflet in diastole (PLAX)
4. Immobility of the posterior mitral leaflet (a similar appearance can be seen in Hyper eosinophilia or Ergot use)
5. Fish-mouth orifice in the short-axis view
6. Increased LA size, with the potential for thrombus formation

Planimetry from the parasternal short-axis view[4,10]

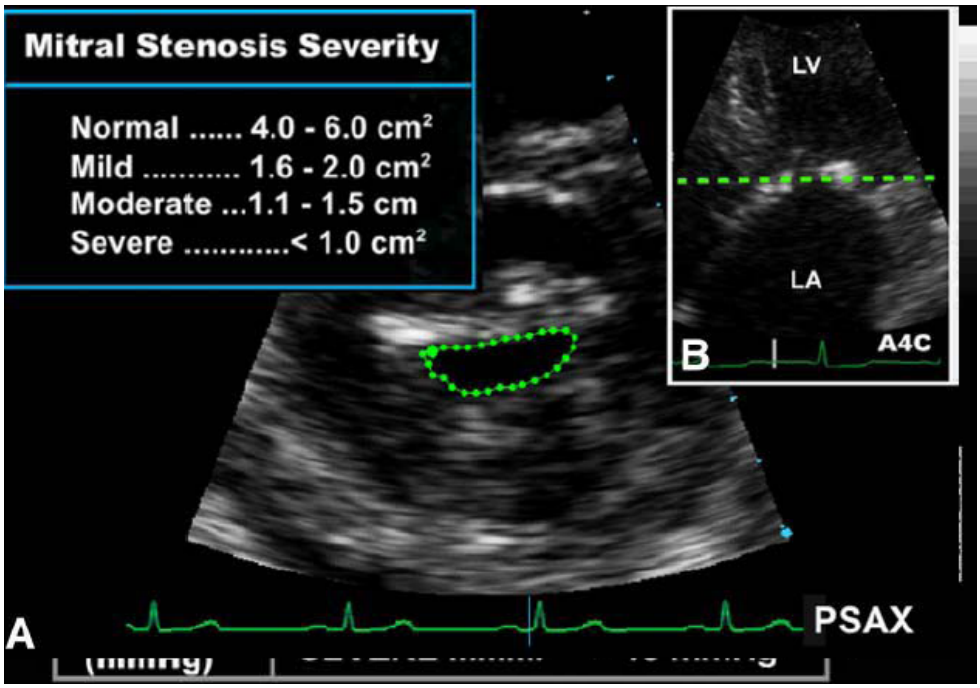


Figure .6

The mitral valve area can be measured with planimetry from the parasternal short-axis view (Figure.), which may be difficult in patients who have heavy calcification or who previously had commissurotomy. In patients undergoing mitral balloon valvuloplasty, an echo cardio graphic score[26] based on valve thickness, calcification, mobility, and subvalvular thickening can be used to predict the outcome of the procedure (Table). Patients with an echo cardio graphic score of 8 or less have a more favourable result from mitral balloon valvuloplasty than those with a higher score, but a score higher than 8 does not preclude the option of valvuloplasty .Commissural calcification or fusion is another important determinant of poor outcome after percutaneous valvuloplasty or valvotomy. Calculation of mitral valve area[10] is the most reliable means of determining the severity of mitral stenosis, and several methods in addition to planimetry[4] of the mitral valve are available.

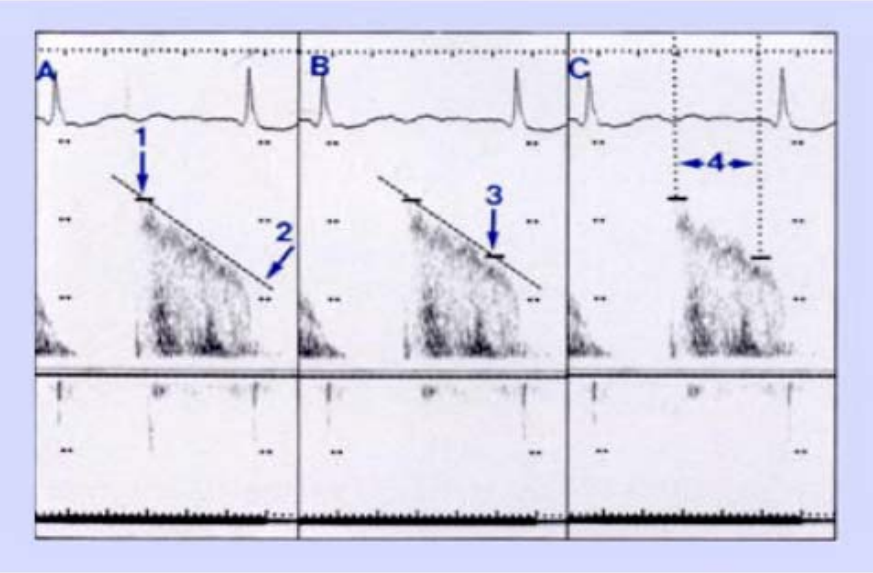
The comprehensive echo-Doppler evaluation of mitral stenosis should include determination of:

1. Mitral valve area (MVA) by the pressure half-time (P1/2T) method: MVA=220/P1/2T
2. Peak mitral diastolic velocity by continuous wave Doppler echocardiography.
3. MVA by the continuity equation and by the PISA method .

Mitral Valve Area by Pressure Half Time :

Formula : MVA =220/P1/2

Pressure Half Time Calculation[1,6]



Pressure Half Time Calculation of Mitral Valve : The P1/2T is the time it takes for the pressure gradient across the MV to decrease by half . The valve area is calculated using the equation, MVA -220/P1/2T where MVA -MV area and P1/2T -pressure half-time. This method correlates well with the invasive measurement of MVA. The concept behind the P1/2T method is as follows: the LV fills when blood from the LA crosses the MV during diastole. As the MV orifice area decreases, blood flow from

the LA to the LV becomes increasingly compromised, and the time required for blood to flow from LA to LV becomes longer. This decreasing valve area is reflected in the length of time required for the pressure gradient across the MV to fall during diastole. The smaller the valve orifice, the longer it takes for the pressure gradient to decrease. In short, MV area is inversely related to the pressure half-time[1] by the formula MVA - 220/P1/2T. It is important to remember that what is measured is the time it takes for the *pressure* to reach one-half of the original pressure. Pressure and velocity are related by the Bernoulli equation (P =4 V2).

There are a number of important caveats to consider when assessing MV area by P1/2T, (as per Masuyama et al[22])

1. Atrial fibrillation. Although the P1/2T is largely independent of heart rate, if the heart rate for any given cycle is markedly elevated, a clear image of the pressure gradient fall may not be visible. Therefore, during atrial fibrillation it may be necessary to evaluate many beats (most labs assess at least 5 and often up to 10 beats).
2. Aortic regurgitation or other conditions that increase left ventricular end-diastolic pressure. Aortic regurgitation can shorten the time it takes for the LV to fill during diastole, as the LV is filled with blood from *both* the LA and the aorta. Therefore, the P1/2T[6] may be "artificially" decreased leading to an overestimation of the MV area. This overestimation typically becomes clinically relevant in the setting of moderate to severe aortic regurgitation. In other conditions in which left ventricular end-diastolic pressure is elevated, such as restrictive cardio myopathy or ischemic heart disease, the rate of equilibration between LA and LV is increased, and the estimation of MV area by P1/2T can also be falsely increased.

3. Post PTMC- A key assumption for this equation is that the P1/2T measurement is largely independent of LA and left ventricular compliance. After PTMC, this assumption is not valid for approximately 72 hours as the chambers re-equilibrate. Therefore, during that time following mitral valvuloplasty, the P1/2T method is not considered valid.

Estimation of the Severity of Stenosis with Doppler [21,22]

Three major technical requirements that must be satisfied if Doppler is to be used for this purpose :

First, an adequate "window" into the chest for ultrasound propagation and reception must be found so that well formed Doppler profiles can be recorded.

Second, for the velocity measurement to be accurate, this window must allow orientation of the ultrasound beam so that it is as parallel as possible to flow through the valve.

Third, the high velocities present in the disturbed jet often exceed the Nyquist limit of PW Doppler, so that CW or high pulse repetition frequency Doppler must be used.

Ultrasound to estimate the severity of a valve stenosis is based principally on the fact that such obstructions result in an increase in the velocity of flow. In clinically significant mitral stenosis, the diastolic velocity of mitral flow usually exceeds 1.7 m/s. Thus, CW Doppler[6] is required for the detection of this increased velocity and for recording the full spectral profile. To be noted that there is a relationship between the pressure increase (or gradient) across a valve and the velocity of blood flow across the valve. For any given pressure gradient there is a corresponding increase in velocity, as predicted by the

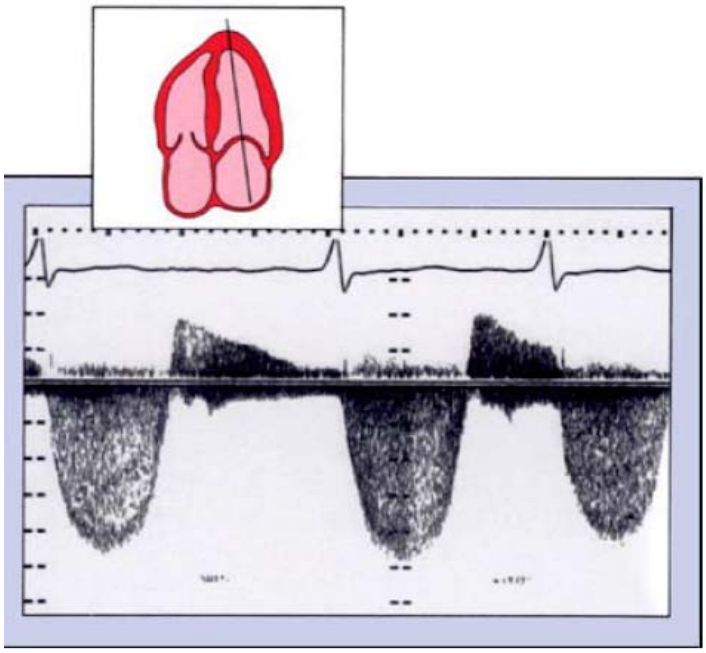
Simplified Bernoulli equation: $\underline{p1\text{-}p2 = 4V^2}$,

Where p1 = pressure distal to obstruction ; p2 = Pressure across the obstruction. As the stenosis becomes more severe, the valve orifice area will become smaller, and the velocity of flow across the orifice will increase as a function of the increased pressure gradient.

Mean Gradient in Mitral Stenosis

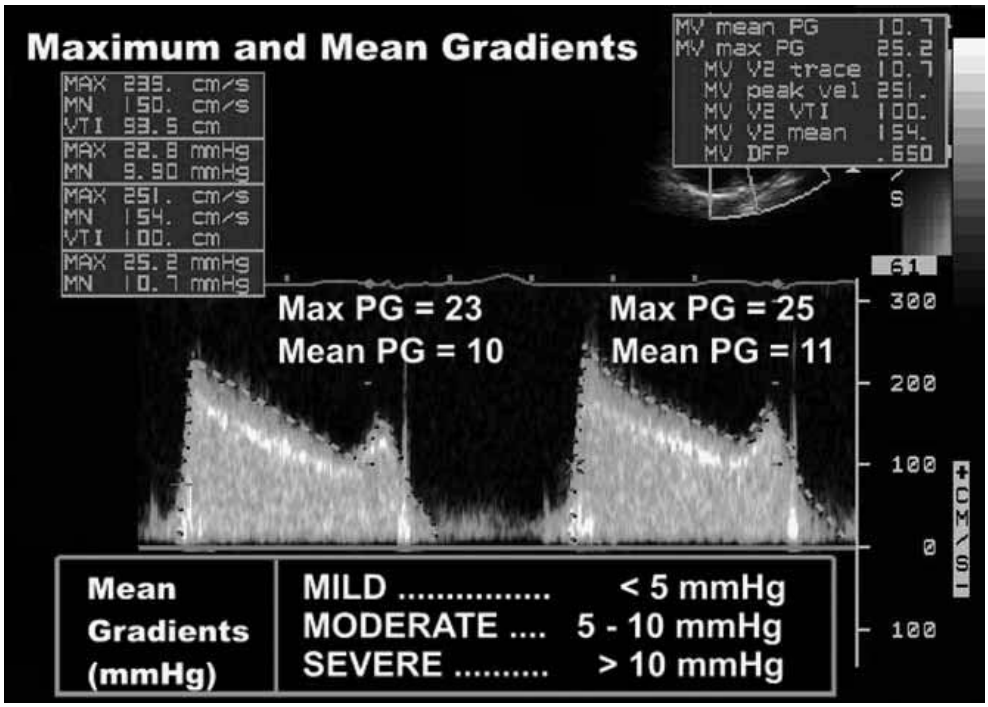
The best window for examination of mitral valve diastolic flow is invariably apical. With the transducer at the cardiac apex, the ultrasound beam should be directed posteriorly and slightly laterally to intercept mitral valve flow. In normal individuals, PW Doppler is adequate for recording mitral valve diastolic flow. Mitral flow is typically laminar and biphasic, peaking in early diastole and rising again with atrial contraction in late diastole. The examination for mitral stenosis is usually much easier and more straightforward than that for aortic stenosis. The typical CW spectral recording of mitral stenosis demonstrates spectral broadening in diastole, with peak flow in early diastole and a progressive but slowed diastole descent. The secondary increase in diastolic velocity due to atrial contraction is absent in patients with atrial fibrillation. A mitral valve gradient is calculated using the modified Bernoulli equation, discussed previously. As with aortic stenosis, the trans mitral pressure gradient may be reported in several ways. Catheterization laboratories usually report the mean gradient. In order to compute a comparable mean gradient for Doppler data, multiple instantaneous peak gradients must be measured during diastole (such as 40-100 ms intervals) and the values averaged. At least 10 well Formed Doppler profiles should be averaged in this manner if the patient is in atrial fibrillation.

Figure. 7 Spectral Doppler



Results published to data have shown excellent agreement between CW Doppler estimates of the mitral valve pressure gradient, using the simplified Bernoulli equation, and simultaneous estimates derived from cardiac catheterization data. However, when the two studies are performed on separate days, the agreement between the two is reduced. This apparent discrepancy derives, in part, from the labile nature of the mitral pressure gradient. The value of this parameter at any particular instant is determined not only by the extent of mitral valve obstruction present but also by the flow across the valve (i.e., cardiac output) and the length of the diastolic filling period, which in turn is determined by the heart rate. Therefore, if the heart rate during catheterization differs from the rate during the Doppler study, the pressure gradients estimated by these two techniques would be expected to differ. In this situation, the higher gradient would be recorded in the study performed at the faster heart rate. The diastolic gradient is relatively low at rest and rises significantly with exercise as heart rate and cardiac output rise **.**

Figure. 8 Severity of Rh.MS



Continuity Equation for Mitral Valve Area Calculation

The MV orifice area can be determined by the continuity principle. According to this principle, flow at any point along a tube is constant (i.e., flow [Q] = VTI1 • A1 = VTI2 • A2, where VTI = velocity time integral at point x and A = area at point x). Therefore, if we know the flow at another point in the "tube" (or heart), e.g., the left ventricular outflow tract (LVOT), and we know the velocity time integral across the MV as measured by CW Doppler, we can solve for the MV area. Specifically, Q = VTILVOT • Area LVOT= VTIMV • Area MV. OrArea MV LVOT/VTIMV. This method correlates well with the invasive assessment of MVA. One key limitation of this method is that if there is regurgitation through The MV or the comparison point (e.g., aortic or pulmonic outflow tract), flow will not be the same at those two points .Therefore, the estimation of the MV area may not be accurate.

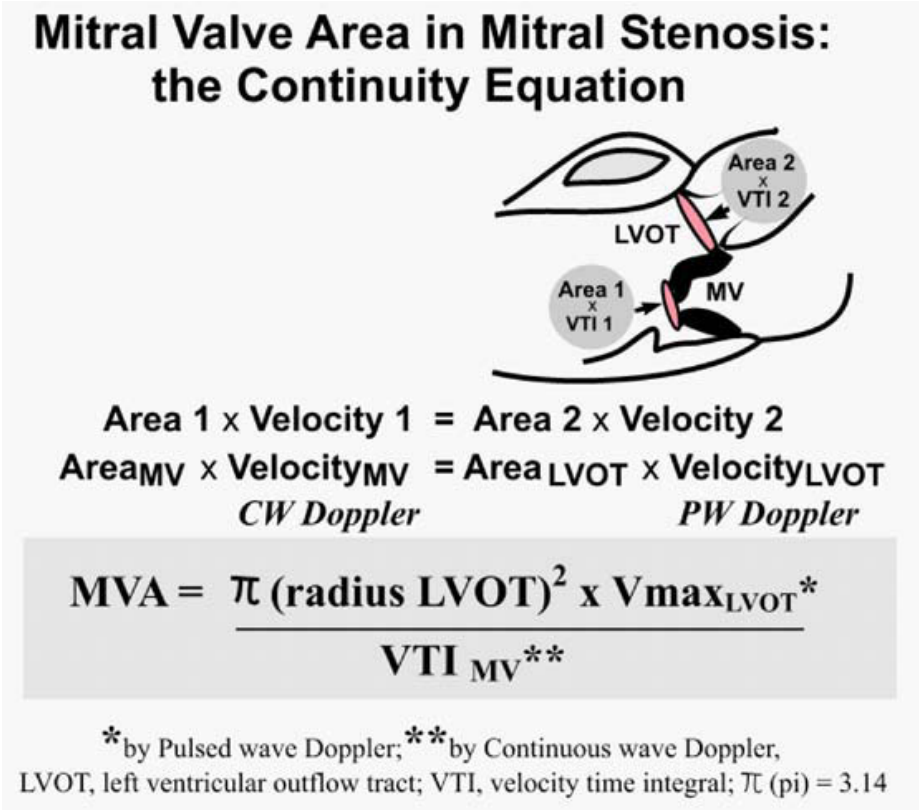
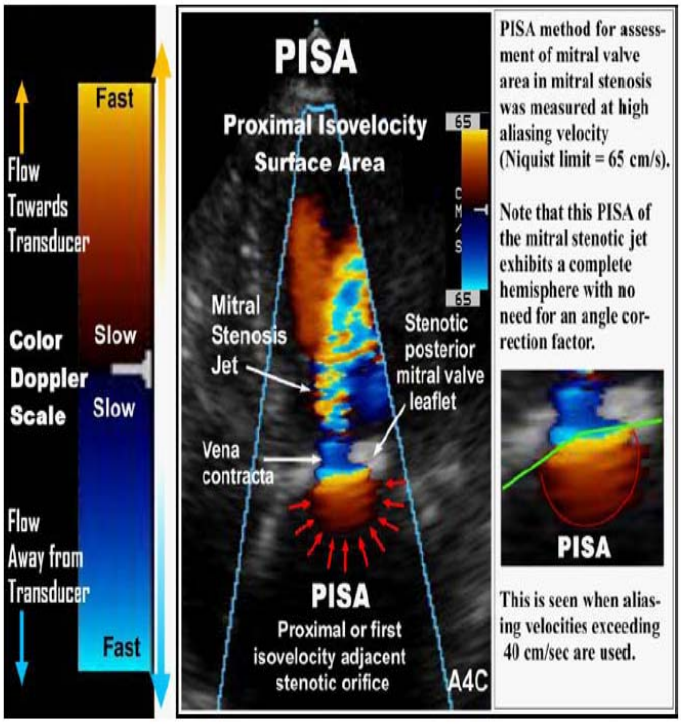


Figure. 9

Figure. 10Proximal Isovelocity Surface Area[7,30]



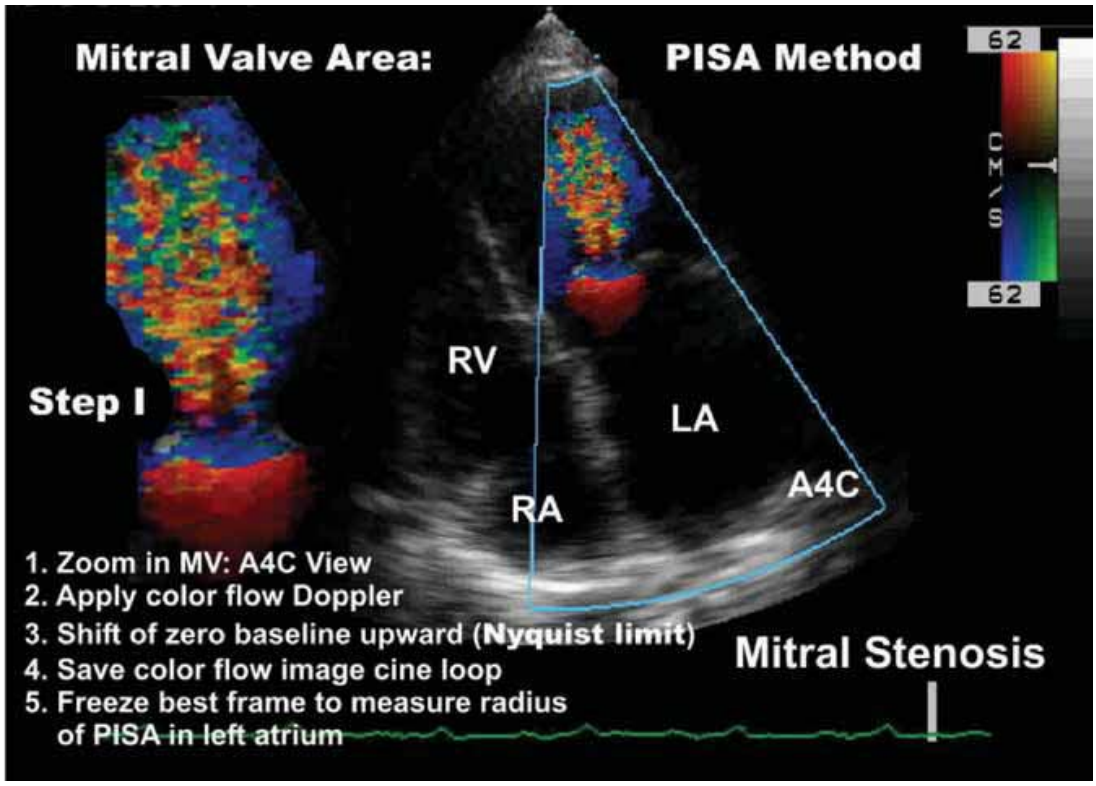


Figure. 11

An extension of the continuity principle is to use the proximal isovelocity surface area to calculate the MVA. Here, the point of "comparison" is changed from the aortic or pulmonic outflow tracts, for example, to the point proximal to the MV in the LA where Color Doppler flow aliases in the shape of a hemisphere. The area at that point is calculated with the equation for the area of a hemisphere, $2\pi r2$, where r is the distance from the point where the color Doppler flow aliases to the orifice of the MV. The velocity at the point where the color Doppler flow aliases is the nyquist limit on the color Doppler setting.

Hence, we have the area and velocity at another point that we may use to solve for the MVA in the continuity equation. (MVA = $2\pi r2$ • aliasing velocity/velocity MV). (Note: an angle correction term may need to be added to this equation in order to account for the influence of the two MV leaflets on the area of the sphere. One benefit of this method is that, in theory, the measurement of MVA[7] is not affected by

coexistent mitral regurgitation as flow across the MV orifice and flow across the aliasing point in the LA will not be significantly different in the presence of mitral regurgitation. However, the proximal isovelocity surface area method[29] is technically challenging and it is not commonly used in many laboratories when evaluating mitral stenosis. Severe mitral stenosis by echocardiography is defined as follows:

1. Resting mean pressure gradient is greater than or equal to 10 mmHg
2. MVA is less than or equal to 1cm^2
3. Pressure half-time is greater than or equal to 220 ms

Increase in trans mitral gradient, LA pressure, and pulmonary artery pressure occurs with the exercise-related increase in cardiac output and heart rate. A mean gradient greater than 15 mmHg with exercise is considered severe mitral stenosis. A dobutamine induced mean mitral pressure gradient greater than or equal to 18 mmHg predicted clinical events with 90 percent accuracy. In patients with mitral stenosis, symptoms have been shown to correlate best with the degree of pulmonary hypertension. In this setting, patients may be treated with a beta blocker to decrease HR response to exercise or with mitral commissurotomy.

Three-Dimensional Echocardiography

Back ground

In 1960s, there were attempts to record and display ultrasound images in 3D format. One of the earliest studies described the acquisition of a series of parallel scans of a human orbit to reconstruct 3D anatomy. Despite the limited technology of the day, the initial studies by Mohr-Kahaly et al[18] demonstrated that complex anatomic structures were ideally displayed using 3D techniques. Concerns about image quality and the computational power needed for storage and reconstruction greatly limited the early application of this methodology. More than a decade later,

investigators began to obtain 3D ultrasound images of the heart. Through the careful tracking of a transducer, a sequence of 2-dimensional (2D) echocardiograms could be recorded, aligned, and reconstructed into a 3D[7]data set. This methodology was limited by the need for offline data processing to create and display the 3D images. In the early 1990s, von Ramm and colleagues developed the first real-time 3D (RT3D) Echo cardio graphic scanner, capable of acquiring volumetric data at frame rates sufficient to depict cardiac motion. More recently, further improvements in design and engineering have led to the commercialization of Live3D echocardiography. This methodology has evolved quickly, and different versions of RT3D imaging are currently available on several platforms.

Methodology

A general approach is to describe cardiac structures using both the ultrasound plane and the viewing perspective. Three orthogonal planes are recommended:

1. The sagittal plane, which corresponds to a vertical, long axis view of the heart.
2. The coronal plane, which corresponds to a 4-chamber view; and
3. The transverse plane, which corresponds to a short-axis view.

Each plane can be viewed from 2 sides, which represent opposite perspectives; for example, the transverse plane, which represents the short-axis view, can be visualized from the perspective of the apex or base; the coronal plane can be viewed from above or below; and the sagittal plane can be viewed from the left or right. The choice of narrow or wide-angle imaging acquisition modes depends on the cardiac structure to be examined. For imaging of the ventricles, it is best to use a wide-angle acquisition in the apical window (4- chamber) so as to include the entire ventricle. For smaller structures, such as the mitral valve, a narrow-angle acquisition may be adequate. The 3D echocardiography[38] technique has contributed significantly to our understanding

of mitral valve function and anatomy. The mitral valve is particularly suited to 3D assessment because of the complex interrelationships among the valve, chordae, papillary muscles, and myocardial walls.

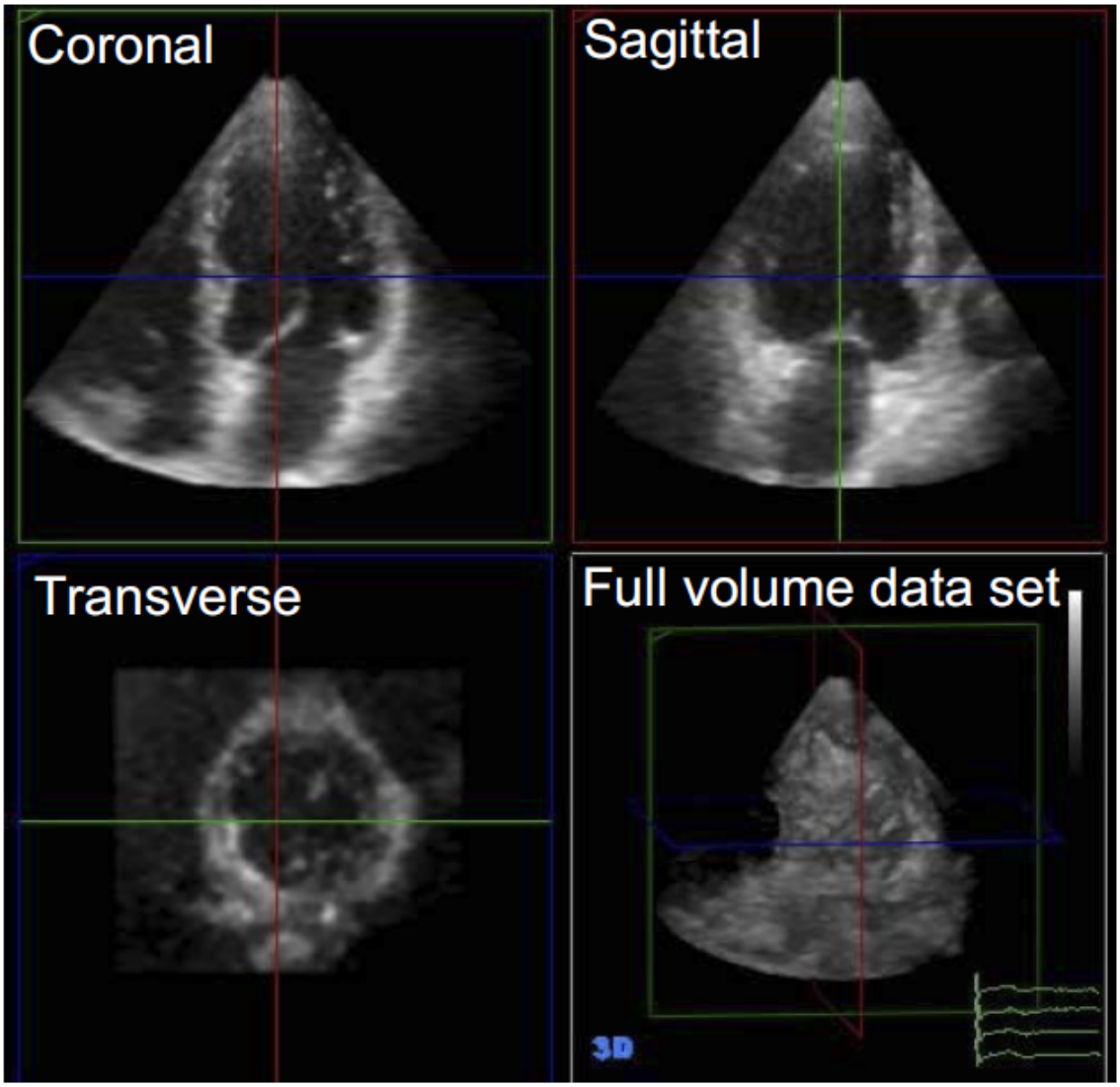


Figure. 12

Using the latest generation scanners as like Philips iE 33 system, we can use Live x Plane imaging to obtain truer 2D planes for analysis. We can perform a single full volume acquisition and crop accordinglyas per details from NC Nanda[39] et al from Card. Clinics 2007. This would not increase significantly the scan time as it requires only 4 heart cycles. The resulting 3D data' set would afford the possibility to

retrospectively achieve any 2D view. 3D imaging[8] of the entire mitral apparatus can be obtained from the trans thoracic or trans esophageal approach[11,40]. Currently available scanners rely on reconstruction techniques from the trans esophageal[11] approach or real-time acquisition of a true three-dimensional data[13] set from the TTE approach[39]. This technique improves the reliability for determination of involvement of chordal structures and further characterization of fibrosis and calcification . At this time, it is limited by its availability and technical complexity of offline reconstruction required in some systems.

Severe Mitral stenosis in Two different Views of 3 D echo[3]

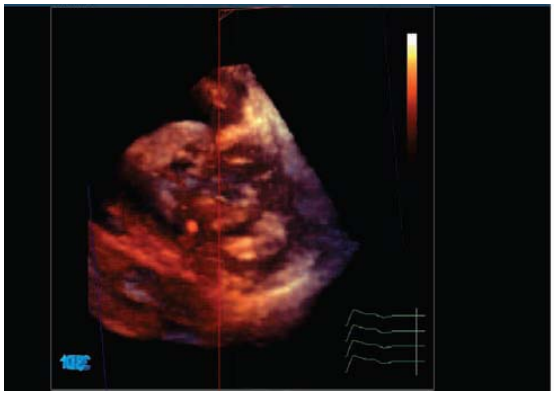


Figure. 13

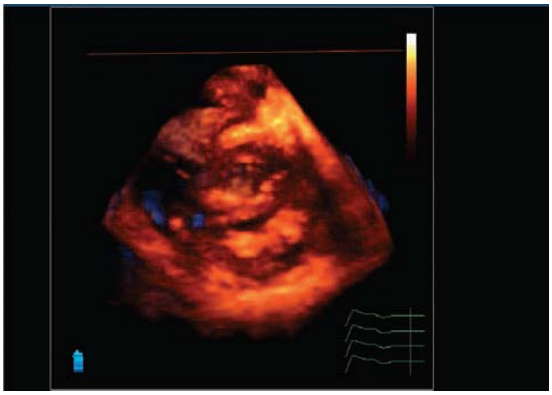


Figure. 14

APPLICATION OF 3D ECHOCARDIOGRAPHY

What is the additional value of 3D echocardiography for assessing mitral stenosis? First, the geometry of the mitral valve can be demonstrated by 3D echocardiography[31], as seen in Figure. Second, as mentioned above, the stenotic valve area[8] can be determined by using unique 3D views. As per Rahimtoola[18] et al, Different 3D echo methods were used to determine the valve area in patients with mitral stenosis. Binder[16] et al. reported real-time volumetric 3D echo[9] data for the estimation of mitral valve area in patients with mitral valve stenosis. In 48 patients with mitral stenosis, MVA was determined by planimetry using volumetric real-time 3D echo[8] and compared to measurements obtained by 2D echo[17] and Doppler pressure

half time[6] (P1/2T). While 2D echo allowed planimetry of the mitral valve in 43 of 48 patients (89%),as per Sugeng[34] et al, calculation of the MVA was possible in all patients when 3D echo was used. Mitral valve area by 3D echo[9] correlated well with MVA by 2D echo (*r* _ 0.93, mean difference, 0.09 cm2) and by P1/2T (*r* _ 0.87, mean difference, 0.16 cm2). Inter observer variability was significantly less for 3D echo[18] than for 2D echo (SD 0.08 cm2 versus SD 0.23 cm2, *p* _ 0.001). In this study, 3D echo reportedly provided accurate and highly reproducible measurements of mitral valve area and can easily be performed from an apical approach.

In another study, a more recent type of real-time 3D echo system was used for planimetry. This was reportedly more accurate than the Gorlin method to measure the valve area although the authors used three classical 2D echo methods (2D planimetry, pressure half time, and the PISA method) as the reference method. The authors concluded in this study that we should keep in mind that 3D echo planimetry may be a better reference method than the Gorlin method to assess the severity of rheumatic mitral stenosis. When 2D methods are used as reference, one may wonder whether 3D echocardiography[9]can be used independently or not in the clinical setting. They personally believe that the combination of conventional 2D echo especially with CW Doppler (for determining pressure gradients and pressure half time) and 3D echo is the best way to accurately assess patho physiology[37] and hemodynamics in patients with MS. As we all know, one should use multiple echo methods to cross-check the values (MVA and pressure gradients).The most striking information by 3D echocardiography[9] is the depth and spatial relationship between two leaflets revealed by rotation of the mitral valve image in motion quoted in Atlas of Heart disease[36]. Not only the severity of the stenosis but also the shape, location, and anatomic abnormalities of the mitral valve leaflets such as heavy calcification, as per Chu JW[34] et al are visualized in a most intuitive way. Also, 3D PISA images give us clear

insight for the location and shape of PISA in MS. As stated before, the 2D PISA method [7,21] assumes a hemispheric shape of PISA to determine MVA, whether the angle is corrected or not. However, the shape of PISA showed an elongated, crescent shape of non-hemispheric geometry. This indicates the absolute necessity of an appropriate correction to use the simple PISA method for determining the MV area with high accuracy.

APPLICATION OF 3D ON PERCUTANEOUS MITRAL COMMISSUROTOMY

Application of 3D echocardiography for commissurotomy has been reported many times. The 3D echocardiography technique has contributed significantly to our understanding of mitral valve function and anatomy. The mitral valve is particularly suited to 3D assessment because of the complex interrelationships among the valve, chordae, papillary muscles, and myocardial walls.

In one of these studies by Hozumi et al[11], multi plane trans esophageal echocardiography (TEE with ECG and respiratory cycle gated image acquisition) was used in 19 patients undergoing balloon mitral commissurotomy . The mitral valve was viewed *en face* as if looking up from the left ventricle. The mean mitral valve area (by pressure half time from the Doppler of the 2D echocardiogram) increased after commissurotomy from 0.86 _0.06 cm2 to 2.07 _0.10 cm2, *p* _ 0.0001. This was similar to the mitral valve areas obtained by planimetry from 3D images. 3D reconstructions showed a complete commissural split in 10 patients and partial splitting in 9 patients. In 3 of the 8 patients who had an increase in the amount of mitral regurgitation, 3D reconstructions were able to detect tears within the valve leaflet. One leaflet tear actually extended up to the mitral valve annulus and was associated with the only

case of severe mitral regurgitation[19]. The authors then concluded that 3D echo cardio graphic reconstruction enabled visualization of the mitral valve so that commissural splitting and leaflet tears not seen on 2D echocardiography became visible. Thanks to recent developments in relatively high quality trans thoracic real-time 3D echocardiography, as per Abascal [19]& colleagues, improvement in measurement of valve area and changes in valve geometry after the balloon commissurotomy was reported. Trans thoracic real-time 3D echo, instead of multi plane TEE 3D reconstruction, could be employed to measure the valve area in patients with rheumatic mitral stenosis who underwent balloon commissurotomy

Invasive Evaluation of Mitral Valve Area

The normal cardiac valve offers little resistance to blood flow. As valvular stenosis develops, there is progressively more resistance to flow, causing a pressure drop (pressure gradient) across the valve. At any stenotic orifice size, greater flow across the orifice yields a greater pressure gradient.

In 1951, the Gorlins[13] introduced an invasive method to calculate the effective orifice area of a stenotic valve using fundamental hydraulics. To calculate the stenotic valve area, the output in liters per minute that flows across the valve needs to be determined. For the mitral and tricuspid valves that means the flow in diastole only. For the mitral and tricuspid valves, the flow in diastole can be determined by multiplying the diastolic filling period (DFP) (seconds per beat) and the heart rate (beats per minute) and dividing the result by the

cardiac output (CO)(ml per minute).

$$\text{Area} = \frac{\text{Flow}}{\text{Constant} \times 44.3 \times \sqrt{\text{Mean gradient}}}$$

or

$$\frac{CO/(DFP \text{ or } SEP)(HR)}{Constant \times 44.3 \times \sqrt{\text{Mean gradient}}}$$

Since the pressure drop or, gradient is a square root function, Since the pressure drop or doubling the cardiac output results in a quadrupling of the gradient. Note that at any particular cardiac output the valve gradient doubling the cardiac output results in a quadrupling of the gradient. Note that at any particular cardiac output the valve gradient increases exponentially as the valve area drops from 1.0 cm^2 to 0.8 cm^2 to 0.6 cm^2, etc. This latter has particular relevance when an intervention such as commissurotomy is performed. There is a marked difference in the valve gradient increasing the valve area from 0.6 to 0.8 cm^2 compared to increasing the valve area the same 0.2 cm^2 increment, from 0.8 to 1.0 cm^2, for instance. The valve area measured by the Gorlin [13]formula is subject to multiple potentials errors. Foremost is the difficulty in determining the cardiac output accurately. In low output states the effective orifice area is often calculated to be smaller than actual area..An alternative to the Gorlin formula is the simplified **Hakki method** It is based on the fact that the DFP times the heart rate produced a number about the same as the Gorlin[20]

$$\text{Valve area} = \frac{\text{Cardiac output (lit/minute)}}{\sqrt{\text{Mean pressure gradient}}}$$

Decision making Regarding intervention

Medical management plays only a minor role in alleviating symptoms in moderate and severe mitral stenosis. Therapy is predominantly directed at increasing the effective mitral orifice area, by open surgical commissurotomy, percutaneous balloon valvotomy, or mitral valve replacement. Once a decision has been made that the severity of mitral stenosis warrants intervention, two-dimensional

echocardiography plays a valuable role in determining the most appropriate interventional or surgical technique. As a general rule, valves with heavy degrees of calcification, chordal shortening and fibrosis, and prominent subvalvar involvement, are not good candidates for either surgical or interventional correction. A mitral valve score has been proposed to further characterize and stratify the degree to which the valve is anatomically compromised. The components of the score are leaflet thickening, leaflet mobility, calcification, and subvalvular involvement. There is a direct relationship between the valve score and the likelihood of successful balloon valvotomy, with higher scores mitigating against successful intervention. In general, calcification and subvalvular involvement represent a disproportionate contribution to the likelihood of technical failure at the time of balloon valvotomy. Individuals with a mitral valve score of 8 typically are excellent candidates for balloon valvotomy, and those with scores greater than 12 are less likely to have a satisfactory result. The issue of balloon valvotomy and intra procedural monitoring success of this procedure with trans esophageal echo is established, which deals with monitoring of operative and interventional procedures. Although percutaneous intervention began with coronary angioplasty and other interventional tools , the concept of treating diseased heart valves began soon thereafter. The initial thrust was to open stenotic pulmonic, mitral, and aortic valves via balloon valvulo plasty for which the basic techniques and equipment have changed little over the last two decades. More recently, there has been a renewed interest in this area as exciting new therapies for percutaneous treatment of mitral regurgitation and percutaneous replacement of pulmonic and aortic valves have entered clinical testing.

PERCUTANEOUS TRANSVENOUS MITRAL COMMISSUROTOMY

Percutaneous mitral commissurotomy is an important therapeutic tool in treating rheumatic mitral stenosis. In third world or developing countries where rheumatic heart disease remains prevalent, percutaneous mitral valvuloplasty is the treatment of choice for treating patients with mitral stenosis**.**

Mechanisms

Percutaneous mitral valvuloplasty is more appropriately called percutaneous mitral commissurotomy because the balloon dilatation improves the valve orifice by separating the fused mitral commissures. As shown by echocardiographic, fluoroscopic, and anatomic studies, the expanding balloon splits fused commissures in the same manner as a surgical commissurotomy.

Patient Selection

Patients should be selected for percutaneous mitral commissurotmy based on both clinical and anatomic factors. In most cases they should be symptomatic, and mitral valve area as measured by echocardiography and hemodynamics should be <1.5 cm^2. Unlike for valve surgery, the presence of pulmonary hypertension or abnormal left ventricular function is not a contraindication. Patients with anatomically suitable valves who have developed re stenosis (commissural refusion) after prior surgical or balloon commissurotomy can also undergo percutaneous mitral commissurotomy with results almost as good as previously untreated patients Although the procedure can be performed in patients of almost any age, the best clinical results are observed in younger patients, with less predictable long-term results occurring in patients older than 70 years, who are more likely to have deformed and calcified valves. Percutaneous mitral commissurotomy is a particularly

valuable tool in treating the symptomatic pregnant woman with critical mitral stenosis. It can also be a lifesaving emergency procedure in the patient with mitral stenosis and refractory pulmonary edema or cardiogenic shock . Asymptomatic patients should be considered for percutaneous mitral commissurotomy when they develop pulmonary hypertension or new-onset atrial fibrillation. A pulmonary artery peak systolic pressure >50 mm Hg at rest or 60 mm Hg with exercise in an otherwise asymptomatic patient represents disease severity that has reached the point where percutaneous commissurotomy should be considered . New atrial fibrillation is less clear an indication but should be considered, especially in patients with Mitral morphology well suited for PTMC.

Contraindications

Although the procedure can be performed at higher risk with thrombus localized to the left atrial appendage, thrombus within the left atrium itself is a contraindication to this procedure. Moderate or severe (2+ on a scale of 0 to 4, determined angiographically) Mitral regurgitation is also a contraindication to percutaneous mitral commissurotomy. Patients with mitral stenosis and aortic or tricuspid valve lesions that require cardiac surgery should be referred for surgery. Concomitant coronary disease can be treated with PCI in conjunction with commissurotomy when the coronary anatomy is suitable. This can be done in one session or staged, with the more clinically severe lesion treated first. Patients with significant valve deformity and echo cardio graphic scores >8 should not be excluded a priori from consideration for percutaneous mitral commissurotomy. There is no absolute contraindication to percutaneous mitral commissurotomy in patients with higher echo cardio graphic scores, but patients with echo cardio graphic scores >8 require an individualized approach. The valve into the left ventricle. The two balloons

are then inflated simultaneously across the mitral valve When properly performed, the double-balloon technique results in excellent improvement in mitral valve area . Multiple studies have shown no significant difference in hemodynamic results (mitral valve gradient or mitral valve area) post procedure between the double-balloon technique and the Inoue balloon system.

An adaptation of the double-balloon technique uses a monorail approach to deliver two balloons across the mitral valve over a single guide wire. The first valvuloplasty balloon with a short monorail segment is passed over the wire across the mitral valve, followed by a second conventional balloon that is then passed over the wire until it is parallel with the first balloon. There are no substantial differences in the mechanism of delivery of force by two balloons using this approach compared with conventional double-wire, double-balloon technique. In the early surgical era of closed heart mitral commissurotomy, a metallic dilator, or commissurotome, was used via a left ventricular apical incision. However, the Inoue balloon technique is faster and less cumbersome and generally requires less fluoroscopy time than these other approaches .The Inoue balloon allows simple progressive upsizing of the balloon without withdrawing the balloon from the left atrium as an important advantage if larger balloon sizes are needed. The Inoue balloon system may, however, result in a slightly higher incidence of mitral regurgitation.

All ante grade approaches begin with the crucial first step of successful trans-septal catheterization. This technique, not only requires successful access to the left atrium, but must also be through the proper part of the atrial septum to allow easy access to the mitral valve. After successful placement of a Mullins-type dilator and sheath into the left atrium and confirmation of its position by a hand injection of contrast, the patient is anti coagulated with heparin. Baseline hemodynamics are then

recorded, confirming the appropriate degree of mitral stenosis. Subsequently, a special solid-core

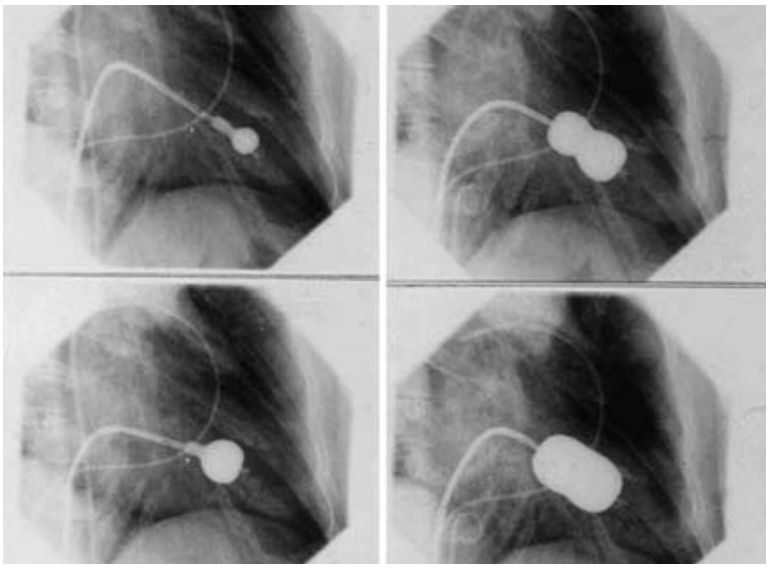


Figure. 15

coiled 0.025-inch guide wire is introduced into the left atrium, and the Mullins sheath dilator system is removed. The femoral vein and inter atrial septum are then dilated with a long 14F dilator over the coiled guide wire within the left atrium. The previously prepared, tested, and now slenderized Inoue balloon is then introduced over the guide wire into the left atrium. The Inoue balloon is made of nylon and rubber micromesh. Owing to the variable elasticity along its length, the balloon inflates in three distinct stages. This allows for stable positioning of the balloon catheter across the mitral valve, as described below. After the slenderized balloon has been positioned within the left atrium, the stretching tube is removed, and a pre shaped stylet is introduced into the Inoue balloon. The distal portion of the balloon is inflated slightly to aid in crossing the valve and to prevent intra chordal passage. By manoeuvring the balloon catheter while rotating and withdrawing the sty let, the balloon tip will move anteriorly and inferiorly toward the mitral orifice. After the balloon catheter is across the mitral orifice, the distal portion of the balloon is inflated

more fully and the catheter is pulled back gently to confirm that the inflated distal portion of the balloon is secure across the mitral valve. As further volume is added to the balloon, the proximal end inflates to lock the valve between the proximal and distal balloon. Inflation to pre calibrated volume then dilates the valve orifice to the corresponding preset size. The sequential filling and positioning of the Inoue balloon is done. It is then allowed to deflate passively before it is withdrawn into the left atrium. The pressure gradient across the mitral valve is measured after each balloon dilatation, and echocardiography may be used to assess the mitral valve area[24], leaflet mobility, and the degree of mitral regurgitation. If the first inflation has not resulted in a satisfactory increase in the mitral valve area, and the degree of mitral regurgitation has not increased, the balloon is then readvanced across the mitral valve and inflation repeated with the balloon diameter increased by 1 or 2 mm by delivery of slightly more of the pre calibrated syringe volume in a stepwise dilatation process that is repeated until the desired result is achieved.

Pre&Post PTMC Mitral Valve area by Planimetry[24]

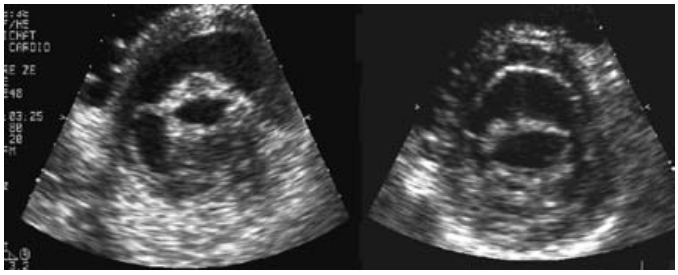


Figure. 16

AIM OF THE STUDY

Our aim was to assess which echo cardio graphic method has the best agreement with the mitral valve area (MVA) invasively evaluated by the Gorlin's formula. We had to evaluate the feasibility and reproducibility of Three-dimensional echocardiography for the estimation of MVA and the Wilkins score in patients with rheumatic mitral stenosis.

MATERIALS & METHODS

STUDY DESIGN:

This study is a comparative study. This study was conducted to describe the various methods of Mitral valve assessment in patients with Rheumatic Mitral Stenosis.

Total no. of patients:

There were totally 50 patients included in this study.

Place of study:

This study was conducted in Government Rajaji Hospital, Madurai. 50 consecutive patients who were attending Cardiology out patients department fulfilling the inclusion criteria were included in this study.

Inclusion Criteria:

Patients with clinical features of Rheumatic Mitral Stenosis (as mentioned in Proforma).

Exclusion Criteria:

All patients with clinical features of Rheumatic Mitral Regurgitation Grade II & above (as mentioned in Proforma)

Consent:

We got informed consent in all our study population. All patients were underwent a proper systematic clinical examination as mentioned in the Proforma immediately after the enrolment. ECG was taken with 12 channel ECG machine. Routine investigations were done. Chest X-ray PA was taken in all patients.

Objectives:

- To analyze the Clinical profile of patients with Rheumatic Mitral Stenosis.
- Mitral valve area to be determined by conventional echo –Doppler methods and by 3D with full volume (Philips IE 33 with matrix array transducer).
- To compare the echo cardio graphic findings with the Invasive Mitral valve assessment (Gorlin 's Formula).
- Mitral score (Wilkin's score) has to be measured.

In the last decade, multiple studies depicted discrepancies between mitral valve orifice area (MVA) measurements obtained with the pressure half-time (P1/2 T) method and invasive methods during the immediate post-percutaneous mitral commissurotomy (PTMC) period. Our aim was to assess the accuracy of 3D full volume Planimetry to measure the MVA in the pre & immediate post-PTMC period. The invasively determined MVA was used as the gold standard.

We hypothesised that since three-dimensional (3D) echocardiography allows a different and superior evaluation of mitral valve apparatus, this technique could increase the ability to perform an accurate MVA planimetry immediately after a PTMC. The use of the new Trans thoracic 3D matrix array probe (Philips, IE 33) allows online 3D rendering of cardiac structures enabling a fast and accurate analysis of cardiac structures.

Non-invasive Evaluation:

A complete echo-Doppler study was performed in all patients using a Philips IE 33 ultrasound machine. Two-dimensional echocardiographic views of the mitral valve were obtained from the parasternal window, and planimetry was performed. Continuous-wave Doppler recordings through the mitral valve were obtained from the apical four-chamber window, and MVA was estimated by using the formula

220/P1/2T. Data required to measure the MVA using the proximal isovelocity surface area (PISA) method were recorded from the apical window. Three cardiac cycles for patients in sinus rhythm and five for patients in atrial fibrillation were recorded, and their results averaged for every patient.

We used the following methods to assess the mitral valve area by 2D echocardiography,

1. **Pressure Half time Method**: The P1/2 T is the time it takes for the pressure gradient across the MV to decrease by half. The valve area is calculated using the equation, MVA -220/P1/2 where MVA -MV area and P1/2 -pressure half-time. This method correlates well with the invasive measurement of MVA.
2. **2D Planimetry**: In our study mitral valve area was measured by planimetry from the parasternal short-axis view Drawbacks noted were as follows it was difficult in patients who have heavy calcification or who previously had commissurotomy. Advantage is that it is not affected by an acute fall in gradient immediately following the procedure.

   Wilkins Score Analysis: The Wilkins score was determined using 2D and 3D echocardiography by an experienced observer at different times. Flexibility, calcification, and subvalvular involvement were assessed. Valvular thickening was not evaluated as it is an objectively measurable parameter.

TABLE -- Echocardiographic Score Used to Predict Outcome of PTMC(Wilkin's Score)[26]

Grade	Mobility	Subvalvular Thickening	Thickening	Calcification
1	Highly mobile valve with only leaflet tips restricted	Minimal thickening just below the mitral leaflets	Leaflets near normal in thickness (4-5 mm)	A single area of increased echo brightness
2	Leaflet mid and base portions have normal mobility	Thickening of chordal structures extending up to one third of the chordal length	Midleaflets normal, considerable thickening of margins (5-8 mm)	Scattered areas of brightness confined to leaflet margins
3	Valve continues to move forward in diastole, mainly from the base	Thickening extending to the distal third of the chords	Thickening extending through the entire leaflet (5-8 mm)	Brightness extending into the midportion of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles	Considerable thickening of all leaflet tissue (>8-10 mm)	Extensive brightness throughout much of the leaflet tissue

3. **PISA Method**: After acquiring the image in A4C view, color flow mapping is applied. The image is zoomed and the baseline is shifted to the Nyquist limit. The time velocity integral is calculated and also the radius of color aliasing velocity in the LA side. Mitral valve area is calculated using the MVA = $2\pi r2$ • aliasing velocity/velocity MV.
4. **3DFull Volume:**[46] Three-dimensional echocardiography was performed immediately after the 2D study. The system scans a 60° x 30° three-dimensional pyramid of data. From different acoustic windows, multiple cardiac cycles of the mitral valve were recorded using the "zoom" mode. Cardiac cycles were also acquired using the "full-volume" mode that consists of the acquisition of a larger single pyramid of data (120° x 60°) recorded during four consecutive cardiac cycles (ECG timed). All images were stored in a magneto optical disk or compact disk and transferred for offline analysis. 3D planimetry was performed "en-face" at the ideal cross-section of the mitral valve during its greatest diastolic opening. The ideal cross-section was defined as the most perpendicular view on the plane with the smallest mitral valve orifice. Gain, time gain control and depth settings were adjusted to provide optimal images of the mitral valve. The slice thickness of the SAX plane was reduced to the smallest possible value, which still permitted visualization of the entire mitral valve circumference (slice thickness 0.6 –1.2 mm). For each beat, MVA was obtained by averaging six measurements. Beat-to-beat variation was analyzed by calculating the coefficient of variation and was compared with the intra observer variability as calculated from six MVA measurements. The smallest MVA[45] was identified from a set of eight parallel short-axis cut planes of the mitral valve between the annulus and the tips of leaflets (para plane echocardiography) and measured by planimetry.

3D Protocol: Live 3D Zoom was done in A3C view. Optimization of image and reduced near field, Time gain compensation in 2D done.

Adjustment of zoom box size and positioning to include entire annulus in the zoom ox was done. Checking of 2D opts and gains in 3D to find the best 3D image was done and the image was acquired. Next, with the A3C view optimization of 2D image reducing the depth and reducing the near field gain and acquiring the image using breath holding was done.

End systolic frame was tagged. MPR`s were aligned. In red quad the image was rotated so that the aortic valve and root can be seen on the left of the image. Blue and red plane were adjusted in such a way that it sits just above mitral annular level. The green plane was aligned in red quad and red in green so that they intersect the mitral valve at mid point. Blue quad was checked so that the red and green planes cut across the short axis of the annular maximum diameter. In the bottom right quad, the model was rotated to match 3D volume operation. The protocol level is also checked to select the level we wish to perform. Number of slices will change automatically. This can be manually changed as well. Next step is to add reference points. Placing annular points at insertion of leaflets into annulus following a left to right workflow is the next vital step.

Editing the nadir and Aortic point at insertion of non-coronary cusp comes next in the 3D protocol. To edit annulus points, the navigate tools in used to move between slices for standard or advanced model. Next, the commissural points are edited on the blue plane watching the red plane changing as a reference to identify and position the commissural points. The leaflets are traced. The coaption and border points are edited next in the blue plane. If not already visualized, the papillary tips are

added in the blue plane until the papillary tips can be seen and labelled antero-lateral and postero-medial.

Proper acquisition of 3D image[44] and application of the aforementioned 3D protocol is the key to the assessment of mitral valve area.

Invasive Evaluation: Invasive hemodynamic evaluation was performed within 24 h of the echo cardiographic[1] recordings. Using the catheter-based data and the Gorlin's equation, the MVA was obtained. Cardiac output was determined. Left ventricle and left atrium pressure tracings were recorded simultaneously by using a 6F pig-tail catheter and a conveniently placed percutaneous trans septal catheter. Planimetry of the area between left atrium and left ventricle pressure tracings was averaged for five beats.

Trans-septal Puncture:

Classically, trans-septal catheterization is performed only from the right femoral vein, although the technique for a trans jugular approach has also been described. Our study was done exclusively using the trans femoral approach. For the femoral approach we used a 70-cm curved Brocken borough needle, which tapers from 18 gauge to 21 gauge at the tip. The needle is introduced via a matching Brocken borough catheter or 8F Mullins sheath and dilator combination that has been inserted to the superior vena cava over a flexible 0.032-inch, 145-cm J guidewire. Once the wire has been removed and the catheter has been flushed, the Brocken borough needle is advanced through the catheter, with an obturator protruding slightly beyond the tip of the needle to avoid abrasion or puncture of the catheter wall during needle advancement. During needle advancement, it is essential to allow the needle and its direction indicator to rotate freely so that it may follow the curves of the catheter and venous structures; the hub of the needle should never be grasped and

rotated at this point. The progress of the needle tip is monitored fluoroscopically, looking for any sign of perforation of the catheter by the needle. The stylet is then removed at the diaphragm, and the needle hub is connected to a pressure manifold, using a stopcock with a short length of tubing, and is carefully flushed. Under continuous fluoroscopic and pressure monitoring, the needle and catheter are then held in constant relationship as they are withdrawn slowly, using both hands. The direction indicator is firmly controlled with the right hand and used to rotate the needle clockwise during this withdrawal from the superior vena cava until the arrow is oriented posteromedialy (4 o'clock when looking from below). As the tip of the catheter enters the right atrium, it moves slightly rightward (toward the patient's left). The needle and catheter are maintained in their postero medial orientation, and they continue to be withdrawn slowly. As the catheter tip slips over the bulge of the ascending aorta, it again moves rightward to overlie the vertebrae in the anterior projection. Further slow withdrawal maintaining the 4 o'clock orientation will be associated with a third rightward movement as the catheter tip snaps into the fossa ovalis. This is confirmed by the fact that advancement will cause the catheter tip to flex slightly (rather than move back up the atrial septum) if its tip is lodged in the fossa. Clear fluoroscopic evidence of fossa engagement is thus essential to successful trans-septal puncture. Once we are confident that the needle tip is across the inter atrial septum, the needle and catheter are advanced as a unit a short distance into the left atrium, taking care to control their motion so that the protruding needle does not injure left atrial structures. When the catheter is across the atrial septum, the needle is withdrawn and the catheter is double-flushed vigorously and connected to a manifold for pressure recording. We used the formula of the Gorlins who first introduced this invasive method to calculate

the effective orifice area of a stenotic valve using fundamental hydraulics. To calculate the stenotic valve area, the output in liters per minute that flows across the valve needs to be determined. For the mitral and tricuspid valves that means the flow in diastole only. For the mitral and tricuspid valves, the flow in diastole can be determined by multiplying the diastolic filling period (DFP) (seconds per beat) and the heart rate (beats per minute) and dividing the result by the cardiac output (CO)(ml per minute).

$$\text{Area} = \frac{\text{Flow}}{\text{Constant} \times 44.3 \times \sqrt{\text{Mean gradient}}}$$

or

$$\frac{\text{CO/ (DFP or SEP) (HR)}}{\text{Constant} \times 44.3 \times \sqrt{\text{Mean gradient}}}$$

Since the pressure drop or, gradient is a square root function, since the pressure drop or doubling the cardiac output results in a quadrupling of the gradient. Note that at any particular cardiac output the valve gradient doubling the CO results in a quadrupling of the gradient.

After Trans-septal puncture.

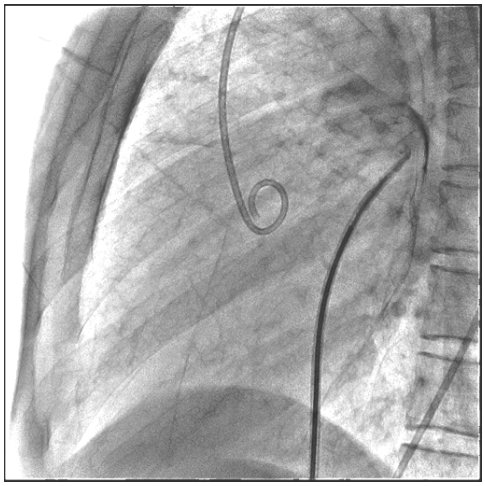


Figure. 17

Balloon Dilatation

After the crucial first step of successful trans-septal catheterization. This technique, requires successful access to the left atrium, but must also be through the proper part of the atrial septum to allow easy access to the mitral valve. After successful placement of a Mullins-type dilator and sheath into the left atrium and confirmation of its position by a hand injection of contrast, the patient is anti coagulated with heparin. Baseline hemodynamics are then recorded, confirming the appropriate degree of mitral stenosis. Subsequently, a special solid-core coiled 0.025-inch guide wire(LA wire) is introduced into the left atrium, and the Mullins sheath dilator system is removed. The femoral vein and inter atrial septum are then dilated with a long 14F dilator over the coiled guide wire within the left atrium. The previously prepared, tested, and now slenderized Accura balloon is then introduced over the guide wire into the left atrium. The Accura balloon is made of nylon and rubber micromesh. Owing to the variable elasticity along its length, the balloon inflates in three distinct stages. This allows for stable positioning of the balloon catheter across the mitral valve. After the slenderized balloon has been positioned within the left atrium, the stretching tube is removed, and a pre shaped stylet is introduced into the Inoue balloon. The distal portion of the balloon is inflated slightly to aid in crossing the valve and to prevent intra chordal passage. By manoeuvring the balloon catheter while rotating and withdrawing the sty let, the balloon tip will move anteriorly and inferiorly toward the mitral orifice. After the balloon catheter is across the mitral orifice, the distal portion of the balloon is inflated more fully and the catheter is pulled back gently to confirm that the inflated distal portion of the balloon is secure across the mitral valve. As further volume is added to the balloon, the proximal end inflates to lock the valve between the proximal and distal balloon.

Inflation to pre calibrated volume then dilates the valve orifice to the corresponding preset size. The sequential filling and positioning of the Inoue balloon is done. It is then allowed to deflate passively before it is withdrawn into the left atrium. The pressure gradient across the mitral valve is measured after each balloon dilatation, and echocardiography may be used to assess the mitral valve area[24], leaflet mobility, and the degree of mitral regurgitation. If the first inflation has not resulted in a satisfactory increase in the mitral valve area, and the degree of mitral regurgitation has not increased, the balloon is then re advanced across the mitral valve and inflation repeated with the balloon diameter increased by 1 or 2 mm by delivery of slightly more of the pre calibrated syringe volume in a stepwise dilatation process that is repeated until the desired result is achieved.

Inflated Accura balloon in Mitral position

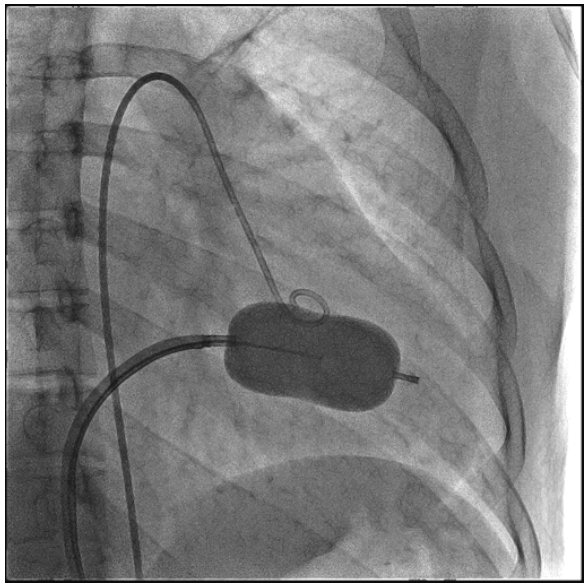
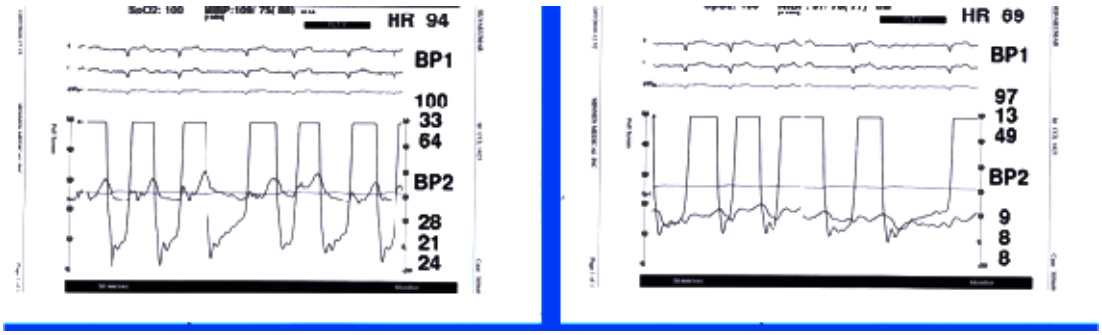


Figure. 18

LA &LV Pressure Tracings :

Pre PTMC **Post PTMC**



Intraobserver Variability: All the recorded images were analyzed offline at different times by an experienced observer. The same images were also analyzed on a different day by the observer.

Statistical Analysis: The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2008)** developed by Centre for Disease Control, Atlanta. Using this software range, frequencies, percentages, means, standard deviations, chi square, 'p' values and Correlation coefficient were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables. A 'p' value less than 0.05 is taken to denote significant relationship. If the correlation coefficient between two variables is more than 0.5, then there exists correlation between those two variables. Regression analysis was done using SPSS –Version 17 Software package.

Figure.19 2D Planimetry

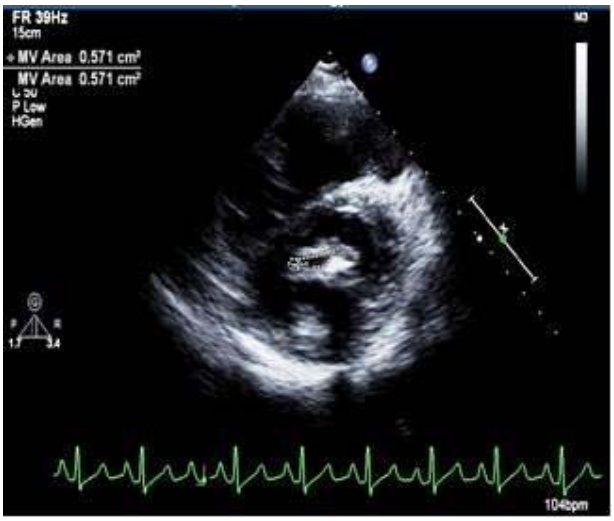


Figure. 20 Spectral Doppler

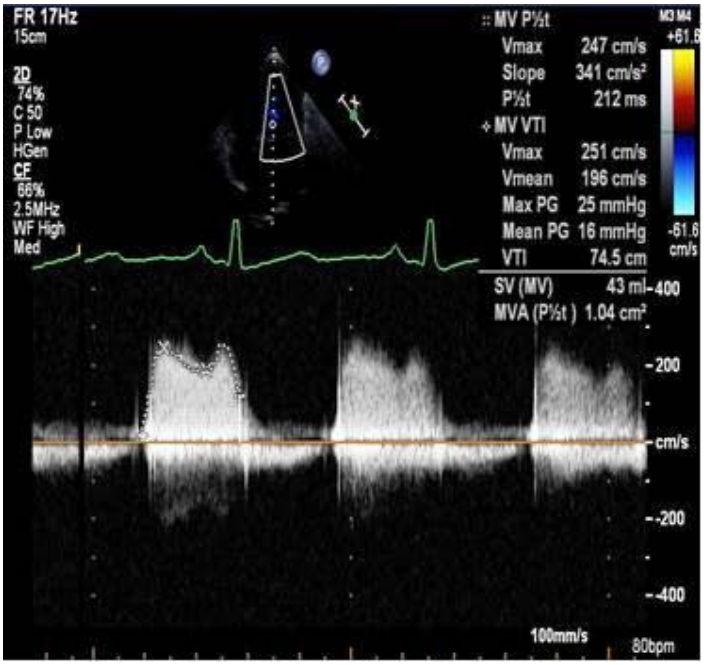


Figure.21 PISA

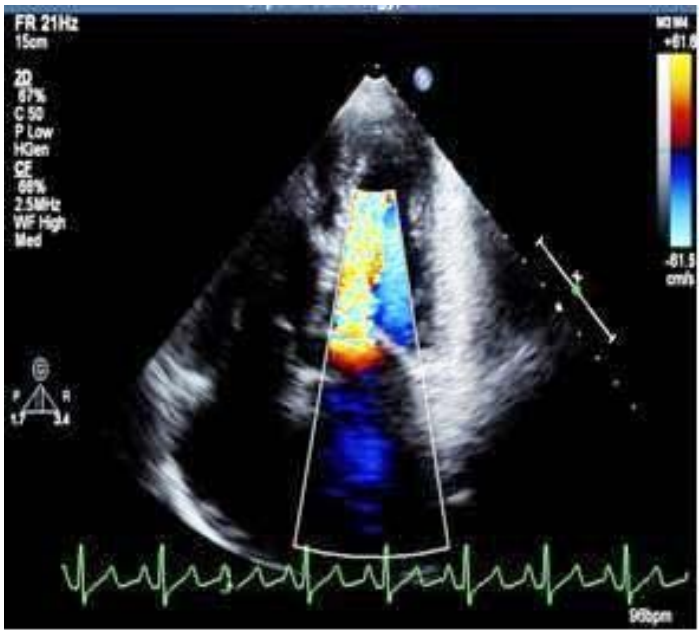
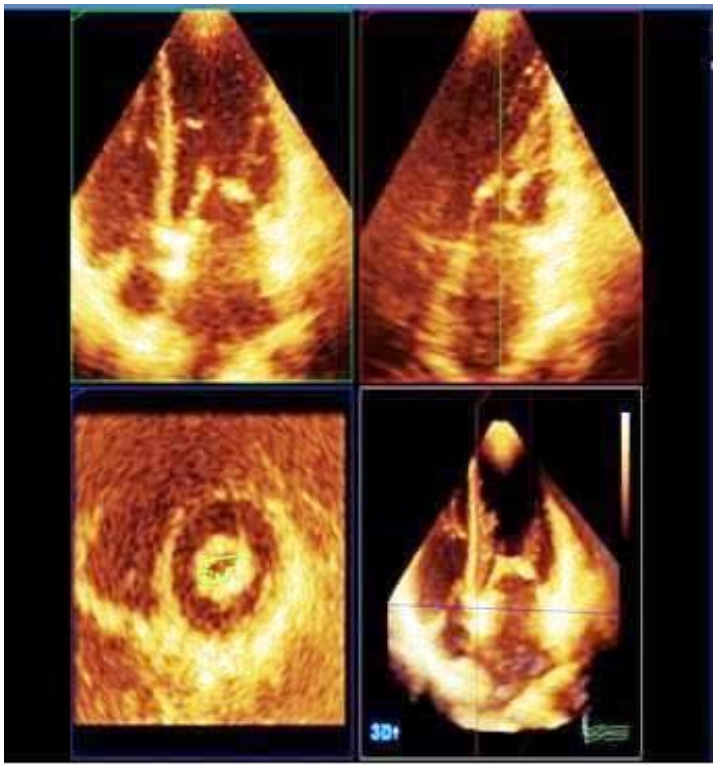


Figure.22 3D Planimetry



RESULTS OF THE STUDY

Fifty consecutive patients with Rheumatic Mitral stenosis comprised our study group (Table.1). There were 32(64%)women (Table2), with a mean age of 32.5± 10 years(Table 3). The Mean LA size was 4.83±0.93 cm(Table 4). Mitral Peak Gradient was 25.7± 6.1mmHg&Mitral Mean Gradient was 15.5 ± 4.2 mmHg (Bar chart). Mitral stenosis was the predominant valve lesion in all of them, but concomitant mitral regurgitation grade I was present in four patients and aortic regurgitation grade I was present in two patients. None of the patients showed aortic stenosis. Regarding the analysis of the tricuspid valve, one of our patient showed organic tricuspid stenosis, but twenty two showed tricuspid regurgitation. The mean TRPG was 41.4 ± 12 mm Hg. Following PTMC, two additional patients were noted to have moderate mitral regurgitation. Forty-two patients were in normal sinus rhythm, eight in atrial fibrillation.

Comparison of non-invasive with invasive Methods MVA determined by the different methods in the pre-PTMC period were: Pressure half-time (P1/2T): 0.86±0.23 cm^2; 2D: 0.85 ± 0.23 cm^2; PISA: 0.68 ± 0.13 cm^2; 3D Full volume Planimetry: 0.63 ± 0.21cm^2; and Gorlin's method: 0.69 ± 0.15 cm^2(Table5).

Regarding the PTMC procedure, the size of the balloon was selected according to the patient's height. Accordingly, the mean size of Accura balloon used was 24.39 ± 1.21. No deep anaesthesia was used for the PTMC.

MVA determined by the different methods in the post-PTMC period were: 2D echo: 2.1± 0.36cm2, 3D Full volume Planimetry: 1.99 ± 0.4 cm2.(Table 7). In the pre-PTMC evaluation, the invasively determined MVA showed a better agreement with 3D Full volume Planimetry results than with P1/2 T or 2D echo results(Table 8). After the PTMC, the higher accuracy of the 3D Full volume Planimetry still remained. Thus, using the invasively determined MVA as the gold standard, 3D Full

volume Planimetry has a better agreement compared to P1/2 T and 2D echo planimetry in both the pre- and post-PTMC periods(Table 9).

The time required to obtain and analyse the 3D Full volume Planimetry images, evaluated in 50 consecutive patients, was 27 ± 5 min. The only used view for 3D Full volume Planimetry was the apical window. Although the presence or absence of atrial fibrillation did not influence the agreement between the measurements with invasive and non-invasive techniques, this fact only affects the time of the echo examination. Among those patients with atrial fibrillation the mean time for the 3D echo evaluation was 32 ± 7 min. 3D Full volume Planimetry is, as with other echo modalities, affected by the quality of the acoustic window. In our study, none of the patients showed bad acoustic window. In 24 patients, the quality was optimal and in the rest considered as excellent.

Comparison of noninvasive with invasive methods.

Mitral valve area determined by the different methods was: pressure half-time (P1/2 T): 0.86±0.23 cm^2; 2D: 0.85 ± 0.23 cm^2; PISA: 0.68 ± 0.13 cm^2; 3D Full volume Planimetry: 0.63 ± 0.21cm^2; and Gorlin's method: 0.69 ± 0.15 cm^2(Table 10). Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables, showed a better agreement when comparing the invasively determined MVA with 3D Full volume Planimetry -determined MVA than when comparing the former with the 2D-, P1/2 T-, and PISA-determined MVA. Agreement between 3D Full volume Planimetry and 2D, P1/2T, and PISA was also evaluated, showing acceptable results. Bar charts displaying differences against average values between traditional non invasive and 3D Full volume Planimetry[42] determined mitral valve area. The best three-dimensional echocardiography[43] method to obtain adequate

images for planimetry was the 3D Full Volume by using zoom method in all patients. The view for 3D planimetry was the apical 3 chamber view.

Valve Score: Evaluation of valve score was different for 2D compared with 3D. The 3D assessment showed the best agreement. The best intra observer agreement when using 2D echo was noted in the evaluation of MV calcification and for 3D in valve flexibility.

In the pre-PTMC evaluation[47], the invasively determined MVA showed a better agreement with Live3D results than with P1/2 T or 2D echo results. After the PTMC, the higher accuracy of the 3D Full volume Planimetry[46] still remained. Thus, using the invasively determined MVA as the gold standard, 3D Full volume Planimetry has a better agreement[42] compared to PISA and 2D echo planimetry in both the pre- and post-PTMC periods. Although PISA also compared favourably with invasive data in the pre-PTMC period, this agreement is lost in the post-PTMC[44] period. The Correlation Coefficient between 3D Full volume Planimetry and Invasive Gorlin's is significant i.e 0.737 which is >0.5 (statistically significant) and the "r" value is 0.956 which is very nearer to 1.000(statistically significant).(Table 9,10)

Table(1): PROFILE OF CASES STUDY

VARIABLE	**VALUE**
Age (yrs)	32.5 ± 10
Sex	Male-18(36%) Female – 32 (64%)
L.A (cms)	4.83 ± 0.93
Mitral PG	25.7± 6.1
Mitral MG	15.5 ± 4.2

Table(2): SEX DISTRIBUTION

Sex	**Cases**	
	No.	**%**
Male	18	36
Female	32	64
Total	50	100

Table(3) : Age Distribution

Age Group (in years)	**Cases**	
	No.	**%**
Upto 20	10	20
21-30	11	22
31-40	18	36
41-50	9	18
Above 50	2	4
Total	50	100
Range Mean S.D.	13-52 yrs 32.5 yrs 10 yrs	

Table (4) : L.A. Size (in cms) [35]

Parameter	**L.A. Size (in cms)**
Range Mean S.D.	3.1-7.9 cms 4.83 cms 0.93 cms

Table(5): Pre – PTMC MV area as measured by various methods

Method	**Pre – PTMC MV area**	**Difference from**		**Correlation with**		**Regression coeff. with**	
		3D-echo	**Gorlins**	**3D-echo**	**Gorlins**	**3 D-echo**	**Gorlins**
PISA	0.68± 0.13	0.05 ± 0.17	-0.072±0.13	**0.5533**	0.4481	0.864	0.676
2D PL	0.85 ± 0.23	-0.22± 0.3	0.16± 0.21	0.1431	0.161	0.126	0.14
PHT	0.86± 0.23	-0.23± 0.28	0.17± 0.18	0.189	0.4092	0.172	0.338

Table(6): Pre PTMC MV Area Gorlin Method

Parameter	**Pre PTMV MV Area by Gorlin Method**
Range	0.5-1
Mean	0.69
S.D.	0.15

Table(7) : Post PTMC MVA and MG as per Gorlin method

Parameter	**Post PTMC**	
	MVA	**MG**
Range	1.4-3.19	3-13
Mean	2	6.57
SD	0.36	2.18

Table (8): Correlation coefficient between the Pre PTMC MV Area measurements by Gorlin Method & Other methods

Method	**Correlation coefficient between Pre PTMV MV Values measured by**	
	Gorlin method	**3DPL**
PISA	0.4481	0.5533
2DPL	0.161	0.1431
PHT	0.4092	0.189
3DPL	0.737	-
Gorlin	-	0.737

Table (9) : Regression coefficient between the Pre PTMC MV Area Measurements by Gorlin Method and Other methods

Method	**Regression coefficient between Pre PTMV MV Values measured by**	
	Gorlin method	**3DPL**
PISA	0.676	0.864
2DPL	0.14	0.126
PHT	0.338	0.172
3DPL	0.956	
Gorlin		0.956

Table(10): Pre – PTMC MV area as measured by various methods

Method	**Pre – PTMC MV area**	**Difference from**		**Correlation with**		**Regression coeff. With**	
		3D-echo	**Gorlins**	**3D-echo**	**Gorlins**	**3 D-echo**	**Gorlins**
PISA	0.68± 0.13	0.05 ± 0.17	-0.072±0.13	**0.5533**	0.4481	0.864	0.676
2D PL	0.85 ± 0.23	-0.22± 0.3	0.16± 0.21	0.1431	0.161	0.126	0.14
PHT	0.86± 0.23	-0.23± 0.28	0.17± 0.18	0.189	0.4092	0.172	0.338
3D PL	0.63± 0.21	-	-0.07 ± 0.13	-	**0.737**	-	**0.956**
GORLIN	0.69± 0.15	**-0.07 ±0.13**	-	**0.737**	-	0.956	-

DISCUSSION

Due to rapid urbanisation and overcrowding, Rheumatic Mitral Stenosis remains an important public health concern in developing countries. PTMC has become the procedure of choice in symptomatic patients when the stenotic mitral valve is not heavily calcified and mitral regurgitation is not significant because it is cost effective and safe. This technique may also be used in patients with less favourable anatomic features, particularly in patients who are considered to be at high surgical risk such as pregnant women, very elderly patients, patients with associated Severe Ischemic heart disease or associated other co morbidities i.e., severe pulmonary, renal, or malignant diseases. The results of PTMC are equivalent to those of surgical, open commissurotomy and both give better results than closed mitral commissurotomy.

Although the Gorlin-derived mitral valve area (MVA) has been used before and after PTMC, echocardiography is of paramount importance in assessing the indication before this procedure, as well as the success and possible complications afterwards. Until recently, MVA was assessed indirectly by the pressure half-time method, or by direct planimetry, with 2D trans thoracic echocardiography[6], by 3D trans thoracic echocardiography[3] or by 3D trans oesophageal echocardiography (TEE). All these methods have their advantages and limitations. Patients with Rh.MS who require an intervention can be easily identified using non-invasive techniques and the results can be predicted by a careful pre-PTMC Doppler echo cardio graphic evaluation. Before the PTMC, the pressure gradient, the mitral valve area, and the severity of valve regurgitation, can be used to evaluate patients reliably. Prior to PTMC, Doppler echo cardio graphic estimation of MVA [6]correlates well with

invasive estimation. Immediately following PTMC, the P1/2 T method has been shown to have a poor agreement with invasive data.

There are various reasons for this inaccuracy including:

1. The development of an atrial septal defect in many patients after PTMC and
2. The P1/2 T method assumes that the left atrial and left ventricular compliances remain stable; this assumption is not valid in the immediate period following PTMC because rapid changes in the left atrial pressure and left ventricular filling occur in this setting, affecting the compliance of both the left atrium and ventricle.

Compared to the P1/2 T method, planimetry (2D or 3D) is not as dependent on haemo dynamic variables (heart rate, cardiac index, cardiac rhythm, left ventricular systolic and diastolic dysfunction, left ventricular and atrial compliance, left ventricular hypertrophy and concomitant valvular disease). Accordingly, planimetry[6] of MVA should be more accurate in the setting of PTMC. Planimetry of mitral valve orifice using 2D echo[6] is a valid method but has its own set of limitations, especially following commissurotomy when the mitral orifice becomes irregular and technically difficult to trace, particularly if calcium is present. Trans thoracic 3D echocardiography [3,15]was the most accurate ultrasound technique for measuring MVA, with a better pre- and post-procedural agreement with the invasively[16] (Gorlin) derived MVA, compared to 2D planimetry and pressure half-time derived MVA. The success rate for 3D echocardiography[14]in 50 consecutive mitral stenosis patients in this centre was 100% for all methods, making Trans thoracic 3D echocardiography a feasible technique, with an acceptable acquisition and analysis time of approximately 27$\pm$5 min. Post- PTMC, the agreement of 3D full volume Planimetry with the Gorlin-derived MVA was much better, in contrast to 2D planimetry and pressure half-time-

derived MVA[24], which may be due to the hemodynamic and compliance changes affecting the latter, as per our study and Chen CG[23] et al , observed before. Also, compared to conventional 2D planimetry, 3DFull volume Planimetry[14] was superior, especially post- PTMC. In 2D planimetry[23,] mal positioning errors in depth and angle of the ultrasound beam can easily lead to an overestimation of the MVA up to 88%, which is not an acceptable accuracy for patient management.

Furthermore, it was easier and faster to define the image plane with the smallest orifice area, when 3D Full volume planimetry was used, and reproducibility for the Wilkins score was better than for 2D echocardiography. The most striking information by 3D echocardiography[9] in our study is the depth and spatial relationship between two leaflets revealed by rotation of the mitral valve image in motion with 3D full volume Planimetry.

Similarly with PISA, which has better correlation coefficient as comparable to 3D echo, the methodology is cumbersome to follow especially for the beginners, who can do easily the 3D full volume Planimetry. We also inferred that three-dimensional image data sets, by providing the possibility of "computer slicing" to generate equidistant parallel cross sections of the mitral valve independently from physically dictated ultrasonic windows, allow accurate and reproducible measurement of the MVA.

In conclusion, Trans thoracic 3D echocardiography offered visualization of the entire mitral valve apparatus, and allowed en face views of the mitral funnel orifice,[45,47] from which accurate measurements of the MVA can be made pre- PTMC. It was also a very suitable technique for monitoring both the efficacy of the PTMC procedure (commissural splitting, MVA before and after), as well as its complications

(leaflet tearing and mitral regurgitation) with a better accuracy compared to 2D planimetry and pressure half-time derived-MVA 3Dfull volume Planimetry[9] allowed a different and superior evaluation of the mitral valve apparatus, improving the ability to obtain an accurate measurement of the MVA. Restriction of the tips and chordae, during the evolution of the rheumatic mitral valve disease, effectively converts the mitral valve apparatus into a funnel [37]with its restrictive mitral valve orifice being at the tips of the leaflets. Due to the variable geometry of the stenotic mitral valve orifice, correct plane orientation frequently becomes difficult. Minor changes in depth and angle of the ultrasound beam led to an overestimation of the MVA[13] by anywhere from 63% to 88%. 3D echo had already been shown to be useful to optimise the results and prevent the development of significant mitral regurgitation during balloon mitral commissurotomy[13]. The use of the new trans thoracic 3D[11] matrix array probe that allowed 3D Full Volume analysis , allowed fast visualization of the mitral valve apparatus and the acquisition of en face views of the mitral valve from which the accurate measurements of the mitral valve area can be made. This image modality should be routinely used to both monitor the PTMC and obtain accurate MVA measurements[12]. In this study, 3D full volume Planimetry is the most accurate echo cardio graphic technique for measuring MVA. Compared with P1/2T, 2D echo planimetry and PISA, 3D echo planimetry had the best agreement when compared to the invasively derived MVA.

Not only did this occur in the pre-PTMC period but also in the post- PTMC period. Importantly, since manipulation of the 3D full volume Planimetry probe is similar to other clinically used Trans thoracic 2D probes, we do not need a long training period to be versatile with 3D image acquisition. We need to know, that although 3D echo provides a more accurate evaluation of the anatomy of the mitral

valve, as with 2D[28] echo, it is importantly influenced by the quality of the acoustic window. Needless to say that although the new equipment provides better resolution and image quality, a bad acoustic window will lead to a poor analysis of the patient.

STUDY LIMITATIONS

The principal limitation of this study was the use of Gorlin's method as the Gold Standard. The Gorlin's equation[13] is a haemo dynamic method that has recognized limitations, especially in situations when rapid haemo dynamic changes occur, such as the post-PTMC period. Significant mitral regurgitation[5,32] and the presence of an atrial septal defect as per Manga [27]et al, may confound measurements of trans-mitral volume[5] flow; this could explain the slight loss of agreement between 3Dfull volume planimetry and Gorlin estimation of MVA in the post-PTMC period, compared to the pre-PTMC period. The desired gold standard for comparison should be the true pathologic inspection of the valve[14]. True inspection and measurement would allow us to determine which method is the most accurate. Echo studies were performed 24 h before and 24 h after the PTMC; not immediately before and after the procedure. Therefore, exactly the same haemo dynamic situation was not present within the non-invasive and invasive situation. For the calculation of mitral valve area, we have used and compared the P1/2 T. Also, functional data as mitral valve resistance have not been calculated in the present study. Other limitations are the image quality and the echo- Doppler settings which could also influence MVA measurements.

Clinical implications

3D Full volume Planimetry improves the assessment of MVA in the clinical scenarios of early post-PTMC period, when other methods have been shown not to be useful. Thus, 3D Full volume Planimetry is able to replace other non-invasive methods and make invasive evaluation unnecessary in this setting.

CONCLUSION

Trans thoracic 3D echocardiography offered visualization of the entire mitral valve apparatus, and allowed en face views of the mitral funnel orifice, from which accurate measurements of the MVA can be made pre- PTMC. It was also a very suitable technique for monitoring both the efficacy of the PTMC procedure (commissural splitting, MVA before and after), as well as its complications (leaflet tearing and mitral regurgitation) with a better accuracy compared to 2D planimetry and pressure half-time derived-MVA. 3D Full volume Planimetry[9] allowed a different and superior evaluation of the mitral valve apparatus, improving the ability to obtain an accurate measurement of the MVA.

Hence, Trans thoracic 3D Echocardiography is a feasible and accurate technique for measuring MVA in patients with Rheumatic Mitral Stenosis, in Pre, Intra and Post PTMC states compared to the Pressure half-time method, PISA and 2D echo planimetry.

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ABBREVIATIONS

MVA	-	Mitral valve area
Live 3D	-	Three-dimensional Echocardiography
Rh.MS	-	Rheumatic Mitral stenosis
PTMC	-	Percutaneous Transvenous Mitral commissurotomy
P1/2T	-	Pressure Half Time of MV
PISA	-	Proximal IsovelocitySurface area
2D PL	-	Two Dimensional Planimetry
3DPL	-	Three Dimensional Planimetry
NYHA	-	New York Heart Association
PLAX view	-	Parasternal Long axis view
PSAX view	-	Parasternal Short axis view
LV	-	Left Ventricle
LA	-	Left Atrium
CW	-	Continuous wave Doppler
PW	-	Pulse wave Doppler
TRPG	-	Tricuspid Regurgitation Peak Gradient
PG	-	Peak Gradient
MG	-	Mean Gradient
VTI	-	Velocity time integral
LVOT	-	Left Ventricular Outflow Tract
A4C	-	Apical four chamber view
A3C	-	Apical three chamber view
DFP	-	Disatolic filling period
SEP	-	Systolic ejection period
TEE	-	Trans Esophageal Echocardiography
ECG	-	Electrocardiogram

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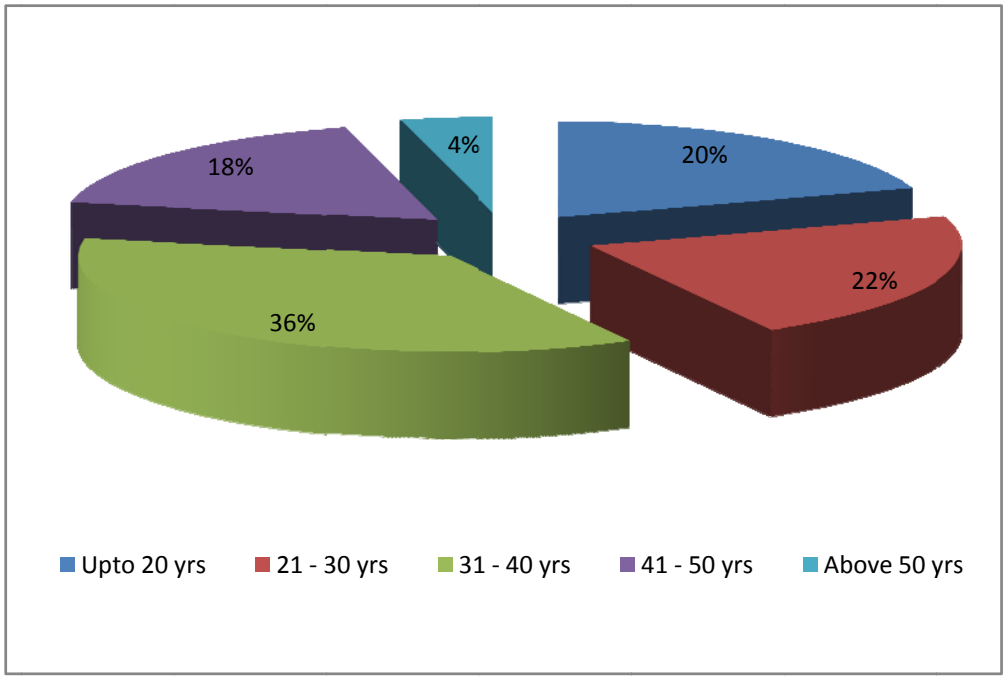

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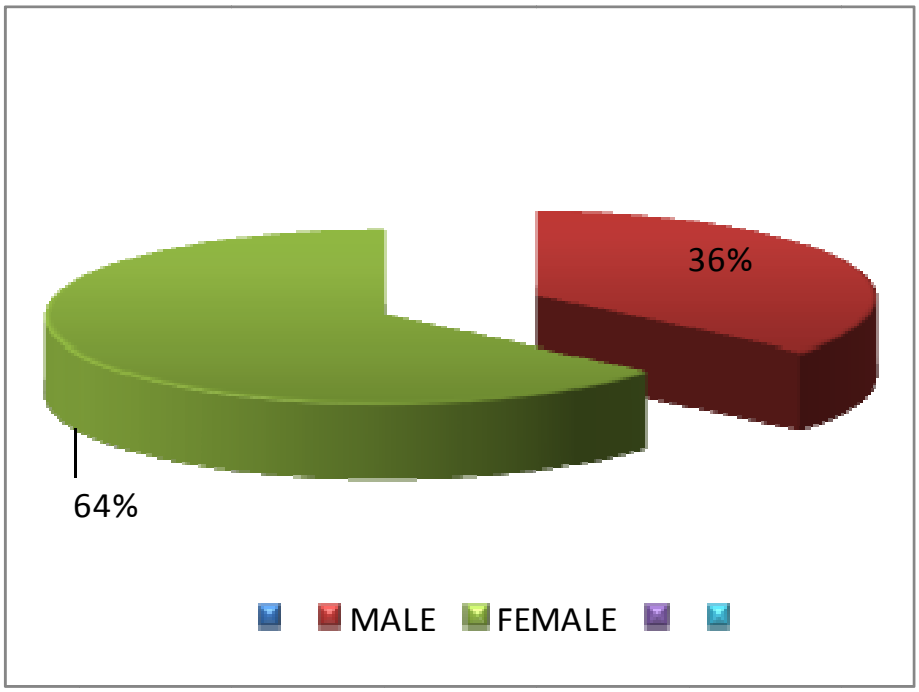

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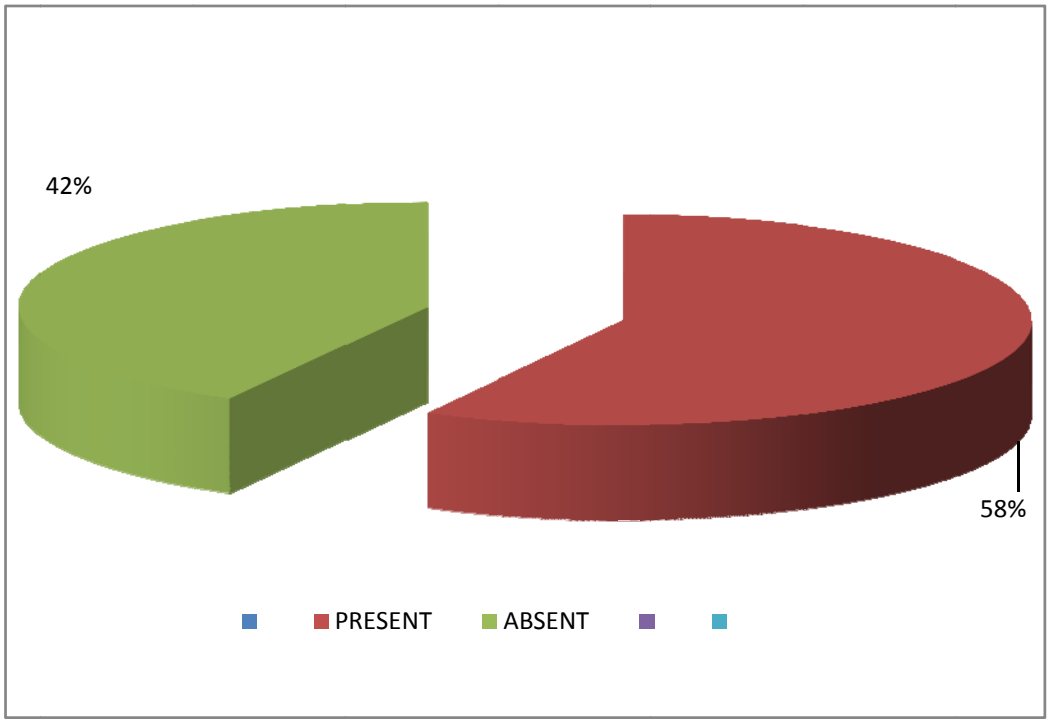

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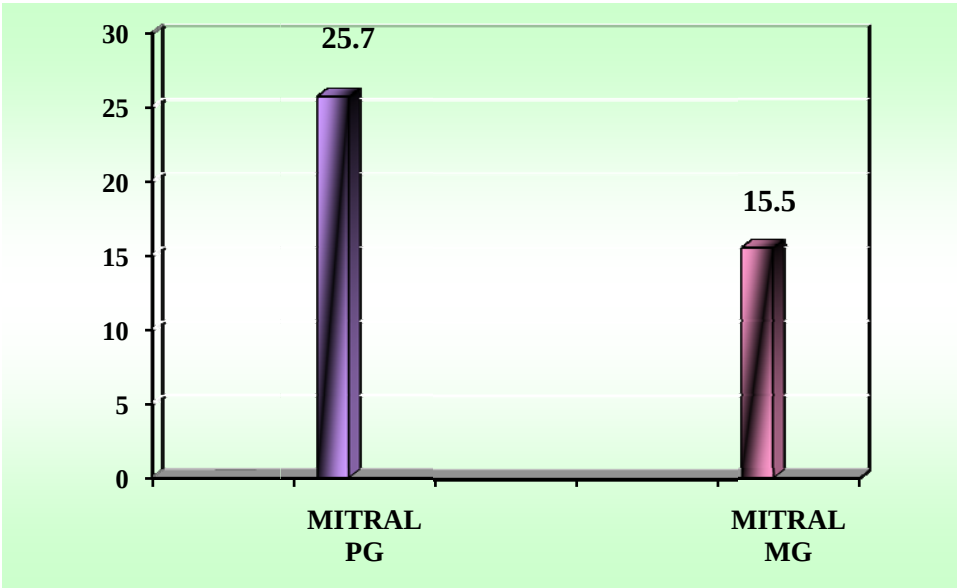

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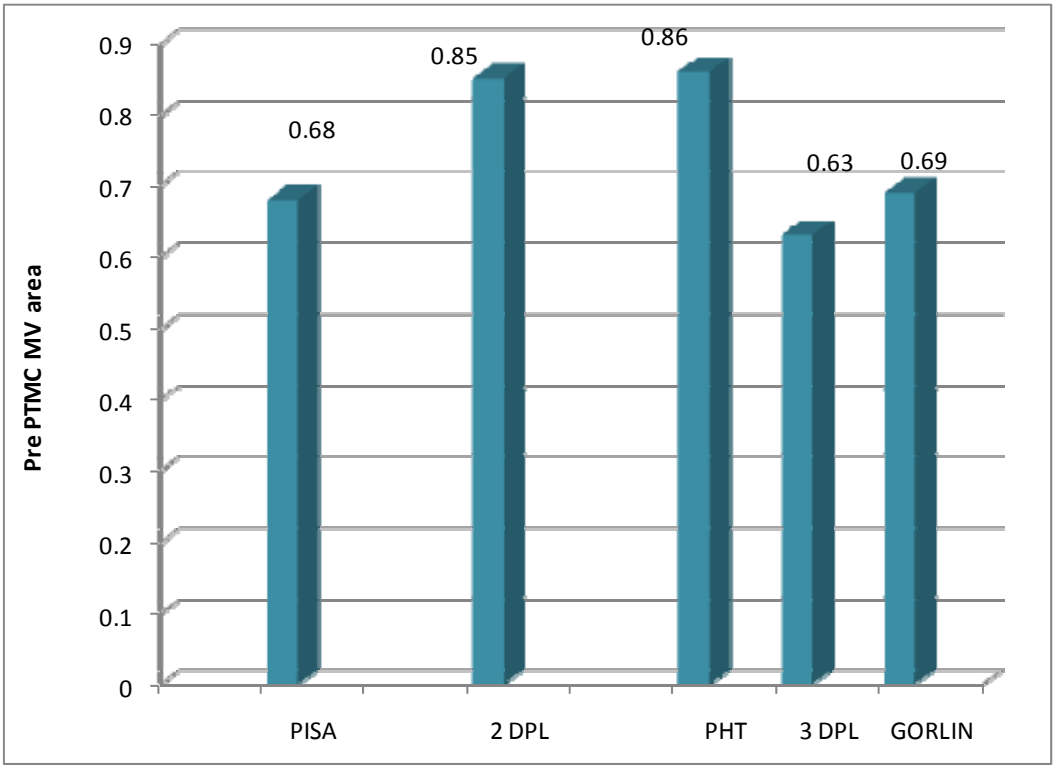
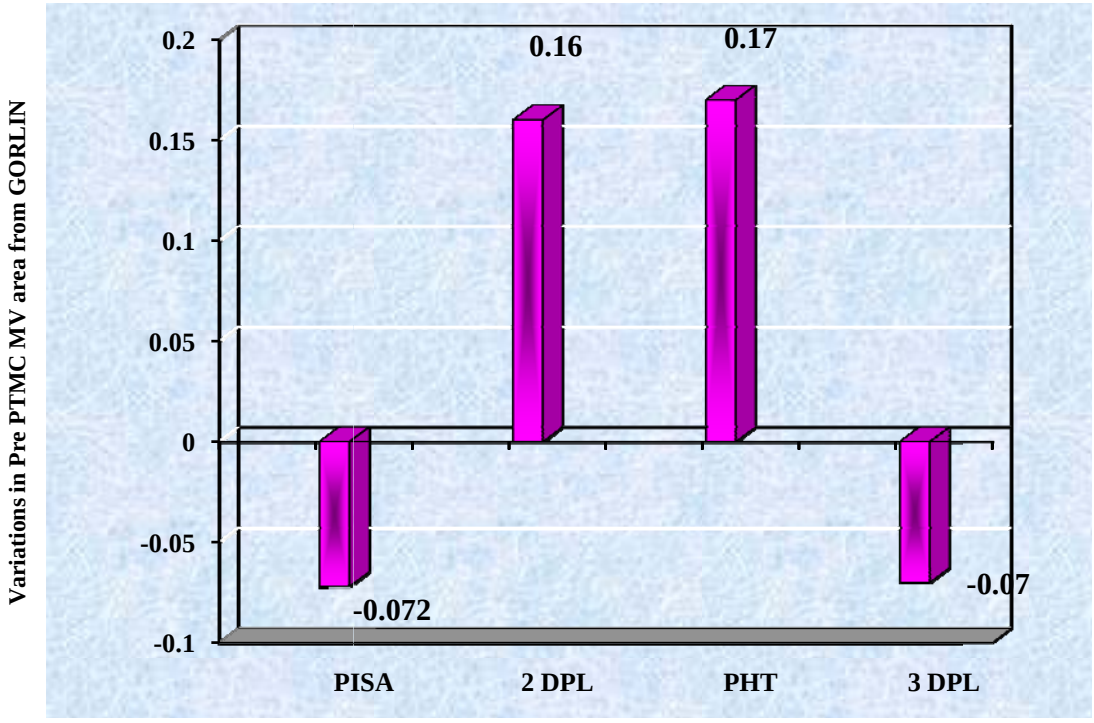
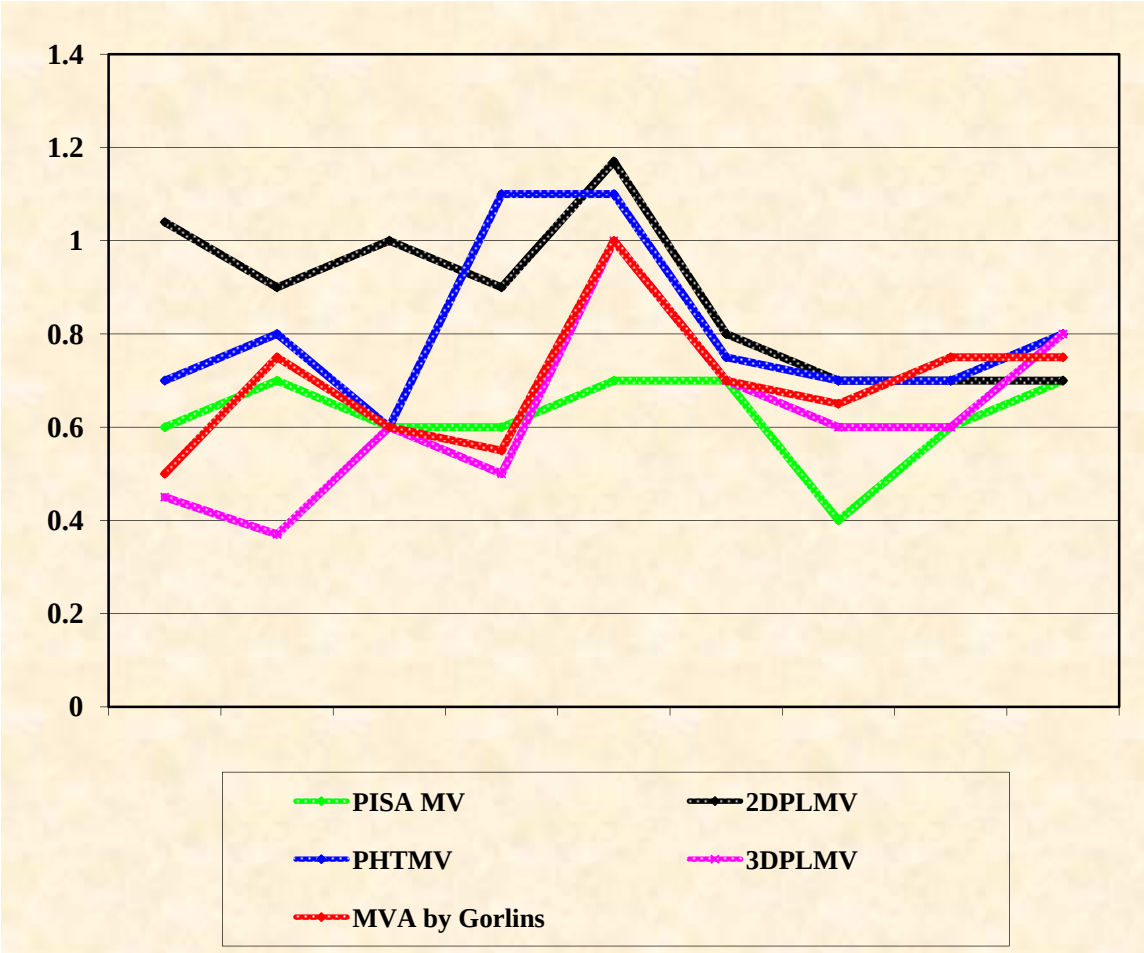


VARIATIONS IN PRE- PTMC MV AREA BETWEEN GORLIN & OTHER METHODS



PRE PTMC MV AREA BY DIFFERENT METHODS



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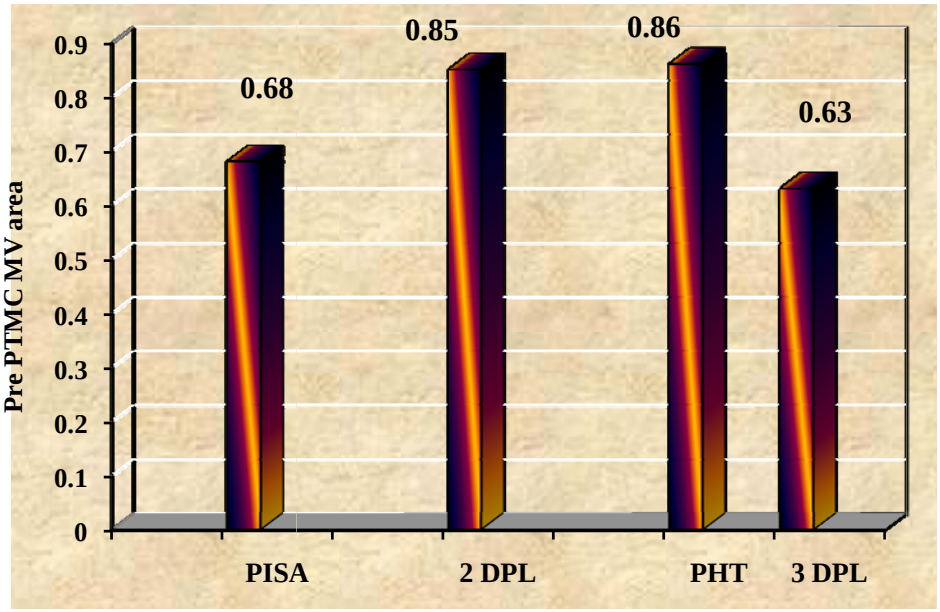

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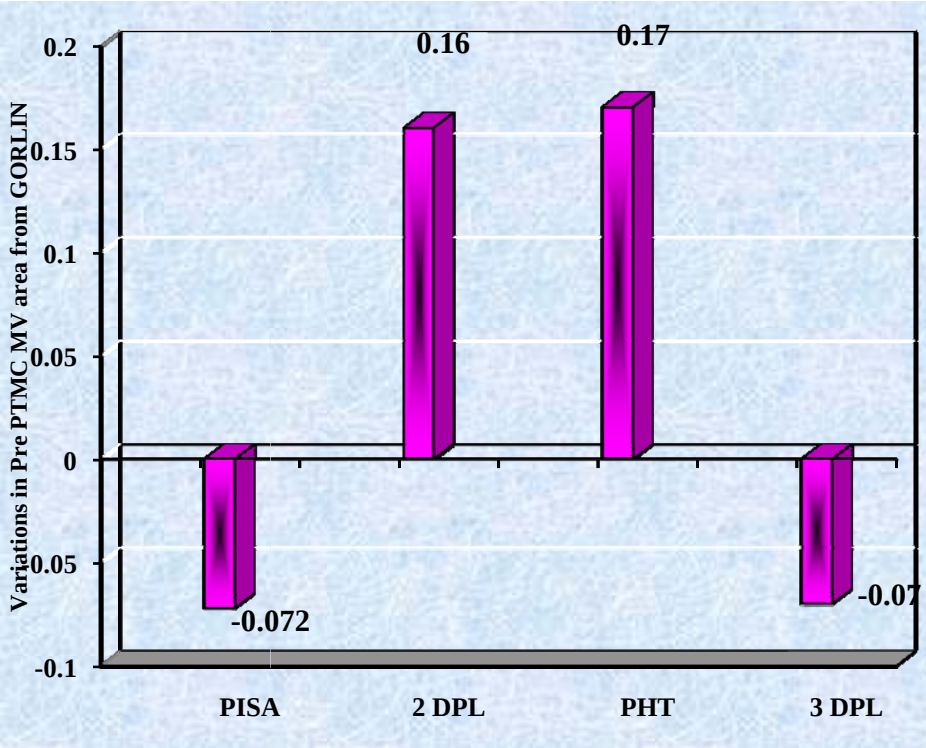
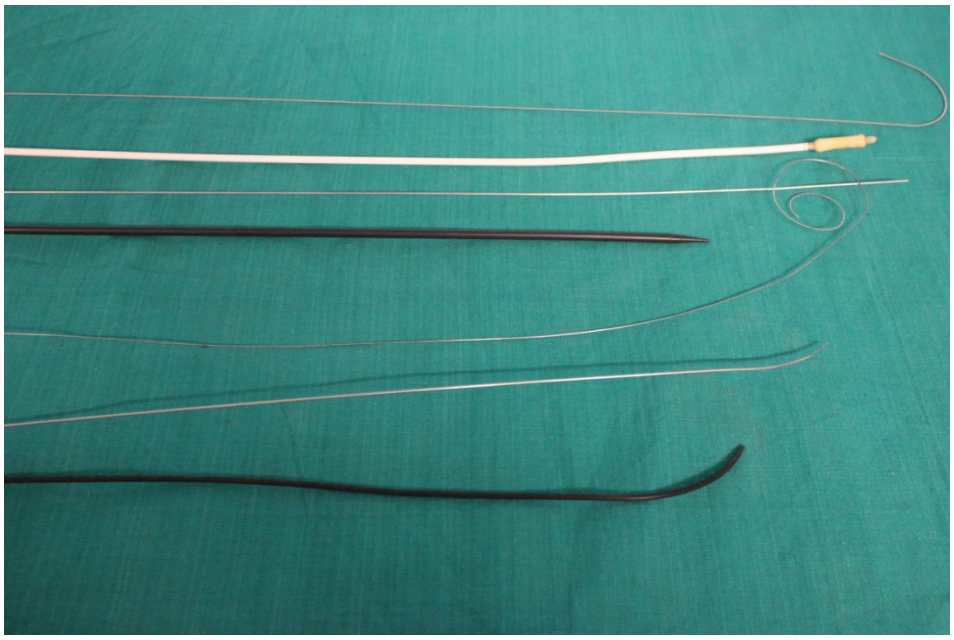
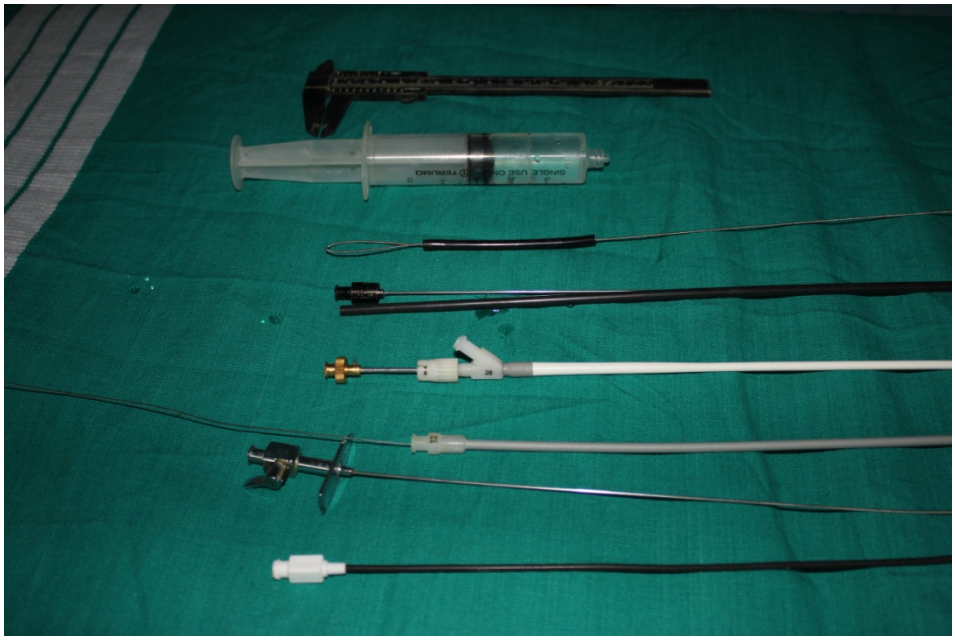
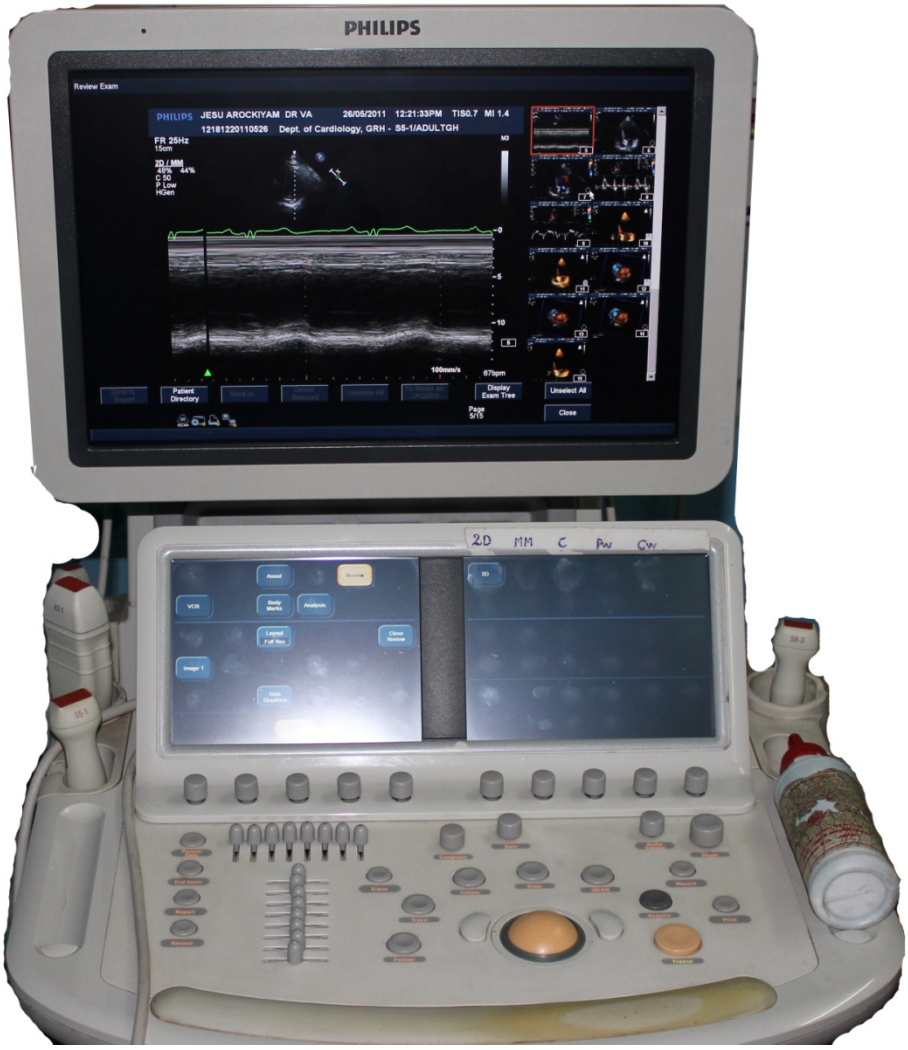


PTMC Set





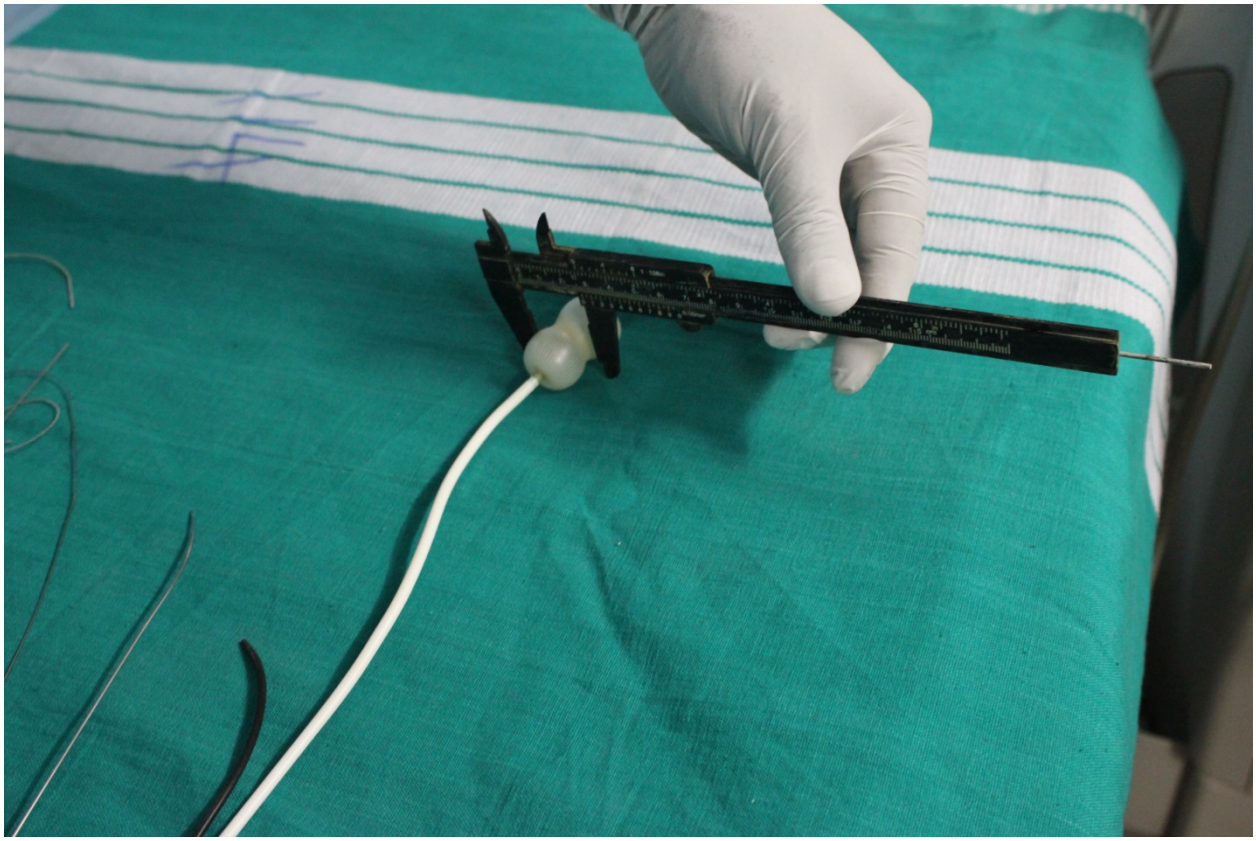
PHILIPS IE 33 ULTRA SOUND SYSTEM



3D Probe

Toshiba Cardiac Cath Lab

Sizing of Accura Balloon



3D Echo Mitral Valve Area Assessment in Rheumatic Mitral Stenosis

Name		Age	Sex
Address		CD No	
Complaints:	Chest pain	Dyspnoea	Giddiness
	Syncope	Palpitation	Cough
	Pedal edema	Fatigue	
Past History:	Diabetes	CVA	Penicillin Prophylaxis
	Rheumatic Fever	Infective Endocarditis	Embolic Phenomena
	Prior Heart Surgery		
Personal History:	Smoking	Alcohol	Exposure to STD
	Socioeconomic Status		
Family History:	Rheumatic Heart Disease		
General Examination:	Pulse	BP	Cyanosis
	Clubbing	Markers of Rh. Fever	Markers of IE
Cardio Vascular System:	JVP	Precordial bulge	Parastemal Lift
	Apex beat	S1 S2	Murmur
Other Systems:	RS	Abdomen	CNS
Investigations:	HB%	Sugar	ESR
12 Lead ECG:	Rate	Rhythm	Axis
	LAE	RVH	LVH
Xray Chest:	LAE	RVH	LVH
ECHO Findings (*Pre-PTMC*)			
General	EF	LA size	Aorta size
Mitral Valve	Annulus	DE Excursion	EF slope
Mitral Flow	'E' grad	'A' grad	
Mitral Gradient	PG	MG	
Scoring	Thickening	Mobility	Calcification
	Subval. Fusion		
Valve Area	2D Planimetry	PHT	3D Planimetry
	MR	AS/AR	TS/TR
	TRPG		
Others	Pericardial Effusion	IAS	LA clot
Disease Severity			

ECHO Findings (*Post-PTMC*)			
General	EF	LA size	RV size
Mitral Valve	Annulus	DE Excursion	EF slope
Mitral Flow	'E' grad	'A' grad	
Mitral Gradient	PG	MG	
Scoring	Thickening	Mobility	Calcification
	Subval. Fusion		
Valve Area	2D Planimetry	PHT	3D Planimetry
	MR	AS/AR	TS/TR
	TRPG		
Others	Pericardial Effusion	IAS	LA clot
Disease Severity			
PTMC Procedure	Date	Balloon size	
	LVEDP-LA grad (*pre*)	LVEDP-LA grad (post)	
	MR grad		

Sno	Name	Age	Sex	CD No	Rh. Fever	LA Size (in CM)	Mitral		TRPG	Pre-PTMC MV Area				Valve Score	2DPL~ 3DPL	PHT ~ 3DPL	PISA ~ 3DPL	MVA by Gorlins	Post PTMC		CCL No/Date
							PG	MG		PISA	2DPL	PHT	3DPL						MVA	MG	
1	**Kumar**	23	M	118051	+	6.69	35	22	42	0.6	1.04	0.7	0.45	7/16	0.59	0.25	0.15	0.50	1.4	8	2320/25.2.11
2	**Kiruthiga**	18	F	170846	+	4.6	30	19	45	0.7	0.7	0.8	0.8	5/16	-0.10	0.00	-0.10	0.75	1.85	5	1394/5.7.10
3	**Mayandi**	25	M	195425	+	4.3	23	13	48	0.6	1	0.6	0.6	5/16	0.40	0.00	0.00	0.60	2.36	5	1395/5.7.10
4	**Moorthy**	40	M	199962		4.5	22	12	27	0.4	0.7	0.7	0.6	6/16	0.10	0.10	-0.20	0.65	2.71	3	1396/5.7.10
5	**Jeyalakshmi**	38	F	212517	+	3.8	23	11	40	0.7	0.9	0.8	0.37	6/16	0.53	0.43	0.33	0.42	1.7	6	1902/12.11.10
6	**Selvakumar**	31	M	213941	+	4.7	22	15	35	0.6	0.7	0.7	0.6	5/16	0.10	0.10	0.00	0.55	2	7	1423/12.7.10
7	**Soma**	50	F	221989		3.4	19	12	28	0.7	1.17	1.1	1	6/16	0.17	0.10	-0.30	1.00	3.19	5	1422/12.7.10
8	**Muniyammal**	20	F	221989	+	3.1	28	20	25	0.6	0.9	1.1	0.5	5/16	0.40	0.60	0.10	0.55	2	7	1421/12.7.10
9	**Kuppayee**	30	F	225391	+	6	38	30	55	0.7	0.9	1.2	0.35	7/16	0.55	0.85	0.35	0.40	1.8	6	2126/6.1.11
10	**Chinnaponnu**	36	F	230602		5.9	23	14	30	0.7	1.2	0.7	0.6	7/16	0.60	0.10	0.10	0.50	2.1	5	2344/2.3.11
11	**Kothandam**	55	M	233436	+	5.8	16	10	27	0.7	0.8	0.9	0.7	8/16	0.10	0.20	0.00	0.65	2.2	4	2219/31.1.11
12	**Ponnuthai**	30	F	293048		6.1	18	13	62	0.7	0.8	0.59	0.45	9/16	0.35	0.14	0.25	0.50	1.75	7	2371/16.3.11
13	**Arammal**	33	F	300389	+	5.7	30	18	32	0.8	0.7	1	0.6	7/16	0.10	0.40	0.20	0.70	1.9	8	2137/10.1.11
14	**Veeramuthu**	33	F	300496	+	5.2	29	18	110	0.6	0.6	0.9	0.4	8/16	0.20	0.50	0.20	0.50	1.6	7	2127/6.1.11
15	**Sumathi**	42	F	301056	+	5.2	21	16	62	0.8	1	0.9	1.2	8/16	-0.20	-0.30	-0.40	1.20	2.52	6	2418/25.3.11
16	**Santhi**	30	F	301543	+	5.3	38	20	50	1	0.5	0.7	1	7/16	-0.50	-0.30	0.00	0.80	2.25	8	2008/10.12.10
17	**Valliappan**	50	M	302832		5.9	30	16	31	0.6	0.4	0.8	0.5	8/16	-0.10	0.30	0.10	0.70	1.6	7	1373/25.6.10
18	**Nagarajan**	33	M	305515	+	5.2	16	11	39	0.8	1.1	1.4	0.4	8/16	0.70	1.00	0.40	0.50	1.7	6	1480/27.7.10
19	**Alagunatchi**	52	M	305642		6.1	32	18	84	0.6	0.7	1	0.36	8/16	0.34	0.64	0.24	0.51	1.7	8	2064/23.12.10
20	**Ponnalagu**	27	F	306634		4.31	30	18	55	0.7	0.61	0.85	0.8	7/16	-0.19	0.05	-0.10	0.70	2.3	7	1915/16.11.10
21	**Nagammal**	41	F	306818		5.56	12	8	26	0.5		0.31	0.47	9/16	-0.47	-0.16	0.03	0.57	1.7	6	1861/2.11.10
22	**Prabavathy**	33	F	306845	+	4.5	27	17	32	0.6	0.9	0.9	0.6	8/16	0.30	0.30	0.00	0.75	2.1	8	1434/15.7.10
23	**Balasubraman**	35	M	306980		5.2	22	15	42	0.7	1.1	1	1	7/16	0.10	0.00	-0.30	1.10	2.4	7	2403/22.3.11
24	**Alamelu**	36	F	307050	+	5.5	35	24	69	0.6	0.4	0.4	0.8	9/16	-0.40	-0.40	-0.20	0.75	1.8	7	2363/15.3.11
25	**Mahalakshmi**	37	F	307247		4.5	33	22	38	0.6	1.2	1.1	0.7	8/16	0.50	0.40	-0.10	1.10	1.8	8	1916/16.11.10
26	**Pappa**	40	F	307407	+	5.6	25	20	57	0.7	0.7	0.71	0.46	8/16	0.24	0.25	0.24	0.65	1.7	7	1761/7/10.10
27	**Ramesh**	52	M	319124	+	4.9	25	15	52	0.6	0.34	0.87	0.35	7/16	-0.01	0.52	0.25	0.55	1.7	6	2047/20.12.10
28	**Rukmini**	35	F	319513		4.5	13	7	48	0.6	0.51	0.8	0.5	9/16	0.01	0.30	0.10	0.75	2	6	2042/18.12.10
29	**Panchavarnam**	33	M	319789	+	3.74	22	15	64	0.3	0.98	0.55	0.5	7/16	0.48	0.05	-0.20	0.65	2	6	2124/6.1.11

30	**Malleswari**	27	F	320132		4.2	22	10	32	0.6	0.6	1.4	0.63	7/16	-0.03	0.77	-0.03	0.63	2.1	8	1478/26.7.10
31	**Dhanamari**	20	F	320412	+	4.3	26	21	46	0.7	0.8	0.75	0.7	8/16	0.10	0.05	0.00	0.72	1.95	9	1688/20.9.10
32	**Kalaimani**	28	M	321970		4.6	14	8	44	0.7	0.8	0.8	0.6	7/16	0.20	0.20	0.10	0.70	1.75	6	1728/29.9.10
33	**Uma selvi**	**20**	F	322144	+	4.1	22	14	28	0.9	1.1	1	0.9	7/16	0.20	0.10	0.00	0.85	1.95	6	1901/12.11.10
34	**Jeyalakshmi**	38	F	322323		4.2	25	15	32	0.7	0.9	1	0.6	8/16	0.30	0.40	0.10	0.70	1.9	7	1902/12.11.10
35	**Natchiappan**	44	M	322371		4.9	23	14	36	0.6	0.9	0.8	0.5	8/16	0.40	0.30	0.10	0.55	2.5	8	1479/26.7.10
36	**Nagaraj**	17	M	323944	+	3.8	26	14	26	0.8	0.7	0.9	0.8	7/16	-0.10	0.10	0.00	0.70	2.42	7	2022/13.12.10
37	**Vanakkili**	37	F	324198		5.6	23	13	28	0.8	1	0.9	0.7	7/16	0.30	0.20	0.10	0.80	2.2	6	1708/24.9.10
38	**Jaffer ali**	40	M	324198	+	4.9	27	16	34	0.7	0.9	0.7	0.6	8/16	0.30	0.10	0.10	0.75	1.9	7	1741/1.10.10
39	**Panchavarnam**	45	F	325662	+	5.1	26	14	26	0.8	0.7	0.8	0.9	7/16	-0.20	-0.10	-0.10	0.75	2.2	6	1778/11.10.10
40	**Vanitha**	27	F	325778	+	5.2	30	16	38	0.6	0.7	0.6	0.6	8/16	0.10	0.00	0.00	0.75	1.76	6	1826/25.10.10
41	**Jeyabharathi**	20	F	326039	+	4.7	22	14	32	0.8	1.1	1	0.6	8/16	0.50	0.40	0.20	0.55	1.95	7	1845/29.10.10
42	**Seetharaman**	26	M	326043		3.9	36	15	48	0.5	0.6	0.5	0.4	8/16	0.20	0.10	0.00	0.50	1.85	6	1869/3.11.10
43	**Pandiammal**	21	F	326263		4.7	30	15	35	0.5	0.9	0.8	0.5	8/16	0.40	0.30	0.00	0.45	2	5	1374/25.6.10
44	**Vallikannu**	38	F	326627	+	3.8	25	14	28	0.7	0.9	1	0.8	7/16	0.10	0.20	-0.10	0.70	1.9	8	2191/21.1.11
45	**Muthumari**	42	M	326744	+	3.9	28	15	28	0.9	1.3	1.2	1	7/16	0.30	0.20	-0.10	1.30	1.9	6	2041/18.12.10
46	**Vijay**	13	M	329277		4.1	28	18	58	0.6	0.8	0.7	0.3	8/16	0.50	0.40	0.30	0.45	1.6	7	1923/19.11.10
47	**Thenmozhi**	17	F	331242	+	4.2	26	14	30	0.9	1.1	1	0.9	7/16	0.20	0.10	0.00	0.80	2.2	8	2339/1.3.11
48	**Alagammal**	23	F	331312		3.5	28	18	28	0.9	1.1	1.2	0.8	7/16	0.30	0.40	0.10	1.00	1.8	7	1909/5.11.10
49	**Saraswathy**	44	F	332492	+	3.9	32	13	30	0.8	0.9	0.8	0.6	8/16	0.30	0.2	0.2	0.55	2	6	1424/13.7.10
50	**Benazir**	18	F	370870		7.9	28	15	30	0.8	1.3	1.1	0.6	8/16	0.70	0.50	0.20	0.55	1.9	7	2209/25.1.11
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3D Echo Mitral Valve Area Assessment in Rheumatic Mitral Stenosis

Name		Age	Sex
Address		CD No	
Complaints:	Chest pain	Dyspnoea	Giddiness
	Syncope	Palpitation	Cough
	Pedal edema	Fatigue	
Past History:	Diabetes	CVA	Penicillin Prophylaxis
	Rheumatic Fever	Infective Endocarditis	Embolic Phenomena
	Prior Heart Surgery		
Personal History:	Smoking	Alcohol	Exposure to STD
	Socioeconomic Status		
Family History:	Rheumatic Heart Disease		
General Examination:	Pulse	BP	Cyanosis
	Clubbing	Markers of Rh. Fever	Markers of IE
Cardio Vascular System:	JVP	Precordial bulge	Parastemal Lift
	Apex beat	S1 S2	Murmur
Other Systems:	RS	Abdomen	CNS
Investigations:	HB%	Sugar	ESR
12 Lead ECG:	Rate	Rhythm	Axis
	LAE	RVH	LVH
Xray Chest:	LAE	RVH	LVH
ECHO Findings (*Pre-PTMC*)			
General	EF	LA size	Aorta size
Mitral Valve	Annulus	DE Excursion	EF slope
Mitral Flow	'E' grad	'A' grad	
Mitral Gradient	PG	MG	
Scoring	Thickening	Mobility	Calcification
	Subval. Fusion		
Valve Area	2D Planimetry	PHT	3D Planimetry
	MR	AS/AR	TS/TR
	TRPG		
Others	Pericardial Effusion	IAS	LA clot
Disease Severity			

ECHO Findings (*Post-PTMC*)			
General	EF	LA size	RV size
Mitral Valve	Annulus	DE Excursion	EF slope
Mitral Flow	'E' grad	'A' grad	
Mitral Gradient	PG	MG	
Scoring	Thickening	Mobility	Calcification
	Subval. Fusion		
Valve Area	2D Planimetry	PHT	3D Planimetry
	MR	AS/AR	TS/TR
	TRPG		
Others	Pericardial Effusion	IAS	LA clot
Disease Severity			
PTMC Procedure	Date	Balloon size	
	LVEDP-LA grad (*pre*)	LVEDP-LA grad (post)	
	MR grad		