SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY

THIRUVANANTHAPURAM, KERALA

PROJECT REPORT

Submitted during the course of
DM Cardiology

Dr. Shyam Sundar Reddy .P
DM Trainee

DEPARTMENT OF CARDIOLOGY

Jan 2008 – Dec 2010

DECLARATION

I , Dr. Shyam Sundar Reddy. P hereby declare that the project in this book were undertaken by me under the supervision of the faculty, Department of Cardiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology.

Thiruvananthapuram
Date :

Dr. Shyam Sundar Reddy P
DM Trainee

Forwarded

The candidate, Dr. Shyam Sundar Reddy. P, has carried out the minimum required procedure.

Thiruvananthapuram
Date :

PROF. Dr. JAGAN MOHAN A THARAKAN
Head of the Department of Cardiology

GENERAL CONTENTS

Report I

Balloon Mitral Valvotomy in Pregnancy

Long term Maternal and fetal outcomes

CONTENTS

Introduction

Rheumatic heart disease still continues to be the commonest heart disease in pregnant women in many countries. Mitral valve disease is the most common form of organic heart disease encountered in India during pregnancy and majority of these patients have mitral stenosis (MS), which contributes significantly to morbidity in pregnancy. Mitral stenosis leads to significant maternal and fetal mortality and morbidity.

Normal pregnancy is associated with a hyperdynamic circulatory state characterized by an increase in blood volume, heart rate and stroke volume, and a decrease in systemic vascular resistance. This increase in cardiac output (usually about 50%) may lead to the unmasking of a previously asymptomatic valve lesion and worsening of already symptomatic lesions. In patients with mitral stenosis, increased blood volume and tachycardia impair left atrial emptying and can lead to a significant rise in the pulmonary veno-capillary pressure.

This hemodynamic stress, along with other factors like anemia, atrial fibrillation and thromboembolism, may precipitate acute pulmonary edema and cardiogenic shock with unacceptable maternal and fetal mortality. In spite of the increase in cardiac output and heart rate occurring after the eighth week of pregnancy, most pregnant women with mitral stenosis can be adequately treated with medical therapy.

However, sometimes an invasive procedure is mandatory to provide a favorable pregnancy course. Surgical mitral valvotomy has been performed in many critically ill pregnant women. Closed or open surgical commissurotomy, however, carries a significant morbidity.

Balloon mitral balloon valvuloplasty (BMV) has been established as an alternative to surgical mitral commissurotomy in the treatment of most patients with symptomatic rheumatic mitral stenosis. Previous reports have demonstrated that BMV can be performed safely during pregnancy in patients with severe mitral stenosis without significant maternal risk or fetal morbidity or mortality. However, at the present time, there are few data on long-term follow-up results after PMV in larger patient populations of pregnant women.

Aims & Objectives

1. To assess the immediate results, complications and long term outcomes in patients undergoing BMV in pregnancy and to compare the data with a group of age matched non pregnant patients undergoing BMV during the same period.

2. To assess the maternal, fetal morbidity and mortality.

3. To assess growth and development of children born out of the pregnancy.

Review of Literature

Although rheumatic heart disease is decreasing worldwide and has become a rare entity complicating in pregnancy in affluent societies, it is still prevalent in the developing world. Rheumatic heart disease remains the most common type of heart disease in pregnant women in many countries, and up to 75% of these patients have mitral stenosis.[1-6]

Pregnancy is a hyper dynamic state characterized by an increase in cardiac output, heart rate, and oxygen consumption. The hemodynamic changes associated with pregnancy may be viewed as normal adaptation to the 15% to 20% increase in oxygen consumption required by the fetus. During the first trimester, blood volume increases up to 60% above values in the non-pregnant state. Cardiac output begins to rise during the first 10 weeks, reaching a peak 30% to 45% above the resting, non-pregnant state by around the 20th week. The increase in cardiac output is associated with a fall in systemic vascular resistance, which results in either a fall or no change in blood pressure, and a widened pulse pressure. Mean heart rate raises, average 10-20 beats/min faster at term; the majority of this increase occurs by the eighth week. These changes are most significant between the 24th and 26th week of gestation and can be explained by an increase in blood volume, the presence of a placental arteriovenous fistula and the hormones produced by the fetus and the placenta.[7, 8]

The normal mitral valve has no pressure gradient across it, and the increased cardiac output of pregnancy is well tolerated. However, in mitral stenosis a gradient develops between the left atrium and ventricle, the magnitude of which depends on the severity of the stenosis and blood flow. Thus at a given severity of mitral stenosis the gradient depends on flow. This flow is equivalent to cardiac output and is determined by heart rate and diastolic filling period. Thus as cardiac output increases during pregnancy, mitral valve flow increases resulting in an increased gradient across the valve. The resultant increase in left atrial pressure causes pulmonary venous hypertension and an increased risk of pulmonary oedema. The pulmonary arterial and right ventricular pressures are elevated and right ventricular failure eventually may ensue. The presence of MS in pregnancy leads not only to low systemic perfusion, but also pulmonary congestion in as many as 25% of patients. [7-11]

During labor and delivery, extra demands are placed on the cardiovascular system. The cardiac response to pain during contractions and the increased amount of blood returning to the heart from the contracting uterus represents an additional 20% rise in cardiac output. The largest fluid shifts occur after delivery when the normal pregnant patient (due to shift of blood from placenta) may experience an increase in cardiac output of up to 65% during the immediate postpartum period.

In addition, tachycardia resulting from pain, stress of labour, inappropriate vasodilatation or prostaglandin-agonist therapy can precipitate pulmonary oedema in women with mitral stenosis.[10.] This occurs as tachycardia is accompanied by a disproportionate reduction of diastolic filling period across the mitral valve and results in elevated left atrial pressures with consequent increased risk of pulmonary oedema. [7-11]

The stenotic mitral valve also limits forward flow and results in tiredness and fainting. Concomitant aortic valve disease, anemia and cardiac arrhythmias may further aggravate the abnormal hemodynamics of mitral stenosis. Atrial irritability may be increased during pregnancy and atrial fibrillation can lead to catastrophic pulmonary edema during late pregnancy as well as thrombus formation and additional risk of cerebrovascular embolization. [7-11]

It is therefore not surprising that mitral stenosis in pregnancy, if not treated appropriately, is associated with a high maternal and perinatal mortality. The rate of maternal death rate is nearly 1% and varies directly with NYHA class (0.4% in class I or II, 6.8% for class III or IV). The highest risk is during the intrapartum and postpartum- period. In the presence of atrial fibrillation maternal mortality is further increased to 14-17%. Similarly, perinatal mortality rates are low in class I-II but increase in class III-IV to 12-31%. [2-6]

Ideally if severe symptomatic MS is diagnosed in a non-pregnant woman desiring pregnancy, it should be treated by percutaneous mitral valvotomy or surgical interventions before the patient conceives. However, for several reasons, this is not always possible. Many of the patients will be detected to have MS during pregnancy for the first time.

MEDICAL THERAPY

During pregnancy patients with severe MS presents a challenging problem because of a high rate of fetal and maternal complications. Although medical treatment is always recommended as first-line therapy, the medications have their own limitations. One must consider also the untoward effects of some drugs in this particular setting. Diuretics are known to decrease placental perfusion. The tachycardia is of sinus origin and is unresponsive to digoxin because the increased sympathetic tone in pregnancy overrides the vagal effect of digoxin.[12, 13]

Beta blockade is often effective in slowing the exertional tachycardia and may allow pregnancy to continue without the need of intervention. However, some patients are not able to tolerate a dose of beta-blocker sufficient to prevent tachycardia due to unacceptable hypotension developing even at low doses and also there have been reported serious adverse effects from beta-blockers on the fetus and neonate.

Beta –blocker therapy in pregnancy has been reported in largely uncontrolled retrospective studies to be associated with fetal growth restriction, bradycardia or hypoglycemia in the newborn infant. However, studies using beta blockers, such as atenolol, indicate that growth restriction is not significant if exposure is minimal and confined to the latter part of pregnancy. [14, 15]

INTERVENTIONS IN MS IN PREGNANCY

Relief of mitral valve obstruction is needed when symptoms persist despite medical therapy. Surgical mitral valvotomy, closed (CMV) or open (OMV) has a significant risk of fetal death. With open surgical commissurotomy performed under general anesthesia and extracorporeal circulation, fetal mortality is between 15 and 33%. Closed commissurotomy carries a lower risk to the fetus, although a 5% to 15% of fetal loss has been associated with this technique as well [16-19]. Zitnik et al have reported a 5% rate of maternal mortality and a 33% of fetal mortality in 22 pregnant women undergoing closed mitral commissurotomy. [18]. Becker et al described one death and 20% of fetal loss in 68 patients after surgical mitral commissurotomy[19]. However, the closed procedure is thought to be less effective than the open commissurotomy and may be more difficult in cases of severe stenosis, calcification, thrombi, or regurgitation.

In addition, surgical experience with closed commissurotomy has declined over the last few decades because the technique has been largely replaced by open commissurotomy or percutaneous techniques in most centers.

BMV IN PREGNANCY

BMV is an effective alternative for the management of patients with mitral stenosis during pregnancy. It was first performed by Inoue. et al in 1984[20] and has proved to be a safe procedure for the mother and the child, with outstanding short term results. The Inoue technique seems to be particularly attractive as fluoroscopy time is short in this method. A comparison of BMV with CMV has shown equivalent efficacy[21].. The hazards of BMV are related to hemodynamic changes during the procedure, radiation hazards and complications like MR and pericardial tamponade.

The hemodynamic risks during BMV are due to hypotension from compression of the inferior vena cava when the mother is in the supine position for a prolonged period and or because of prolonged inflation and deflation of the balloon during dilatation of the mitral valve. This may cause fetal distress, leading to an increased likelihood of delivery by cesarean [22].

Depending upon the dose level, radiation during pregnancy may result in intrauterine growth restriction, microcephaly, leukemia later in life, and other malignancies. Avoidance of BMV during organogenesis, the use of an abdominal shield, and avoidance of left ventricular angiography have significantly reduced the hazard of radiation to mothers and fetuses. The average estimated radiation dose received by the fetus during BMV procedure is 0.2 rad, which is much below the allowed safe dose of 5 rad. Therapeutic abortion is recommended when the fetus is exposed to 10 rad or more [23, 24]. Farhat et al reported no clinical abnormalities in 44 children whose mothers had been submitted to a BMV after a follow-up of 28 months[25]

In pregnant women, some technical problems may be encountered during the BMV procedure. The hypercoagulable state in pregnancy necessitates a quick transseptal puncture, after which heparin can be administered. The gravid uterus may compress and distort the inferior vena cava, making passage of catheters difficult. The elevated diaphragm near term may alter the usual lie of the interatrial septum, making it more horizontal. This is crucial to assess before performing the transseptal puncture. The high cardiac output state of pregnancy produces higher gradients across the MV, which should be kept in mind while assessing the result of BMV during the procedure. [26-29]

BMV can be performed whenever possible, starting from the 12th week of gestation, to avoid the inherent risks of radiation (organogenesis). However, in the presence of unstable clinical conditions, BMV can be performed irrespective of gestational age. Successful BMV during pregnancy should improve the patient's clinical condition, permitting a pregnant woman to return to NYHA functional class I or II as a consequence of improved hemodynamics and MVA. BMV should permit gestation to reach full term, offering the fetus good conditions for adequate intrauterine development and better clinical conditions to the mother until and during delivery.[26-29]

BMV IN PREGNANCY – PROCEDURAL DETAILS

Since BMV in pregnancy is a technically demanding procedure it should be undertaken by operators who are well experienced in BMV procedures. BMV is performed in the fasting state in the catheterization laboratory. Preferably it should be done in the morning hours as the patient is fasting overnight. Surgical and gynecology back-up is usually arranged and ready.

To limit fetal radiation exposure, all patients had their abdomen shielded with lead sheets from the diaphragm to pubic symphysis. There are lead shields available for this purpose. We attach two lead shield sheets together using a adhesive tape and that is used to cover the anterior abdomen. The posterior body is protected by a lead sheet on which the patient will be lying on. The lead shields used for covering the anterior abdomen should be light, so that it will not compress the gravid uterus.

Limiting fluoroscopy should be one of the primary aims in performing a BMV in pregnancy. Fluoroscopy time should be monitored during the procedure & fluoroscopy should be used only when absolutely necessary. Left ventriculography & right heart catheterization are usually avoided to reduce the fluoroscopy time.

All BMV procedures are usually performed under local anesthesia with the Inoue (or similar balloon) technique using the transseptal, anterograde left-sided cardiac approach. Maximum balloon size possible is determined according to the patients' height. (height in centimeters/10 + 10). [30] Right femoral artery and venous access is obtained. Initially a GL or Multipurpose catheter is introduced into the innominate vein and using an exchange length 0.35 guidewire this is exchanged for a Mullins sheath or dilator. It is always good to obtain a blood sample to assess the SVC saturation which will give an idea about the hemodynamic status about the patient.

Then the Brockenbrough septal puncture needle is introduced into the Mullins sheath and simultaneously the assistant puts the pigtail catheter in the non-coronary sinus of the aortic root. Septal puncture is done usually in lateral view and LA is entered. Then left atrial pressure is recorded. Then the pigtail shaped LA wire is introduced into the LA, septum is dilated and Heparin 100 IU/Kg intravenously will be given. Then the balloon is introduced and MV dilatation is done.

Stepwise dilatations of 0.5 mm will be done until a successful result is obtained or any evidence of increasing mitral regurgitation. A successful optimal outcome was defined as a final post-BMV mitral valve area (MVA) of =1.5 cm2 or an increase in MVA of =50% compared with the MVA before BMV in the absence of severe MR [30, 31.]

SHORT & LONG TERM OUTCOMES-BMV IN PREGNANCY

Gupta et al in series of 40 pts with BMV in pregnancy demonstrated a procedural success of 97.5% (39/40). Eleven patients, in whom BMV was performed before 20 weeks of pregnancy, subsequently underwent medical termination of pregnancy uneventfully. 18 patients had a normal delivery, 3 underwent cesarean section for fetal distress, one had a preterm delivery, and there was one stillbirth. Full term delivery data were available in 23 babies-all were healthy without any complications[32].

Nercolini et al in a series of 44 consecutive BMV in pregnant patients showed a procedural success in 95% of the patients with no major complications. Data available form 37 patients showed that 30 pts (81%) reached term and delivered normal infants. 7 pts (18.9%) delivered prematurely, resulting in two fetal deaths; one patient delivered a stillborn. Both studies proved the efficacy and safety of BMV in pregnant patients. [33.]

Mangione et al followed up 23 pts for 5.33 ±6 3.12 years, 91% of them were in FC I and II. Two patients (9%) who had remained in FC III underwent a repeat successful BMV; no further surgery was required. There were no embolic events or death related to the procedure. Echocardiography showed an initial increase in mitral valve area from 1.14± 0.22 cm2 to 2.01± 0.21 cm6 ($P < 0.0001$). During long-term follow-up, it decreased to a mean of 1.75 ± 0.24 cm2 ($P < 0.0001$). 21 children (96%), aged 4.91 ± 2.8 years, showed normal growth and development, and no clinical abnormalities were observed. [34.]

S.N. Routray et al in study of BMV in 40 pregnant women showed that Mitral valve area increased from 0.82 ±0.34 to 1.9 ±0.4 cm2 (P-0.001). One patient had pericardial tamponade. Mean fluoroscopy time was 5.5±3.8 min. There was one stillbirth, no maternal death/abortion/intrauterine growth restriction. All 39 babies were normal at birth. One baby died at 7 months due to pneumonia. On follow up for 36±15 months, all 38 babies maintained normal growth and development without any thyroid disease or malignancy. [36.]

Esteves et al in their study of 71 consecutive pregnant women who underwent BMV- Reported procedural success of 100% with a significant increase in mitral valve area from 0.9±0.2 to 2.0±0.3 cm^2 (p <0.001). At the end of pregnancy, 8% of the patients were in New York Heart Association functional class I or II. At a mean follow-up of 44 ± 31 months, the total event-free survival rate was 54%. The mean gestational age at delivery time was 38± 1 week. Preterm deliveries occurred in 9 patients (13%), including 2 twin pregnancies. The remaining 66 of 75 newborns (88%) had normal weight (mean 2.8 _±0.6 kg) at delivery. At long-term follow-up of 44 ± 31 months after birth, the 66 children exhibited normal growth and development and did not show any clinical abnormalities. [37]

Harikrishnan et.al reported the data of 36 patients from our hospital, showed a procedural success rate of 97.2% (35/36) with no maternal mortality. All patients symptomatically improved & had uneventful deliveries. The children had normal growth and development at a follow up of 2.8± 3.3 years. [38.]

Patients & Methods

Study Population

Cases

The study included 63 pregnant patients who underwent balloon mitral valvuloplasty (BMV) in our center from December 1996 to December 2009. They were considered for BMV because of severe mitral stenosis with uncontrolled symptoms with use of beta-blockers, diuretics, or digoxin, or a combination of these. All subjects gave informed consent, and the risk associated with the procedure was explained, including the risks related to radiation exposure to the fetus. The clinical, echocardiographic and hemodynamic data of these patients were collected from the medical records department and analyzed retrospectively. Clinical and echocardiographic evaluation was performed in all patients at follow-up. All patients were asked to have their deliveries in a tertiary care center where the services of a cardiologist and a neonatologist/pediatrician were available.

After delivery, patients were called for evaluation after 2 months. Details regarding mode of delivery and perinatal events were collected from the patients and analyzed. All patients were contacted by mail and were asked to report for review with the children. The patients underwent detailed clinical and echocardiographic evaluation, and the children had assessment of their growth and development parameters.

Controls

Age and year of procedure matched 121 non pregnant female patients who underwent BMV in our centre from December 1996 to December 2009 were selected randomly and included in the study. The clinical, echocardiographic and hemodynamic data, including follow up data of these patients was collected from the medical records department and analyzed retrospectively. The base line characteristics, immediate results, complications, follow up data were compared between the cases and controls.

Methods

Echocardiographic analysis:

All patients underwent detailed trans-thoracic echocardiographic (TTE) assessment, including 2D imaging, Doppler studies &color flow mapping. The suitability of the valve was assessed by mitral valve morphology, commissural calcium, subvalvar pathology & mitral regurgitation. The scoring system of Wilkins et al was used to classify valves into either high or low risk. [39]

For pregnant group trans-esophageal echocardiography(TEE) was performed only in patients with AF and in those in whom there was a suspicion of left atrial clot on TTE. For the controls TEE was routinely done prior to procedure. TTE was done during BMV, 24 hours after the procedure and at follow up visits.

Balloon mitral valvuloplasty

All procedures were done with standby facility for open- and closed-heart surgical procedures. BMV was performed using the anterograde trans-septal technique as described. [20] We have been using two types of balloon catheters for BMV – the triple lumen Inoue (Toray Inc., Houston, Texas) balloon (up to 1999) and double lumen Accura balloon (Medical concepts, Bangalore, India). Maximum balloon size possible was determined according to patients' height. [30]

Definitions

Procedural success was defined as an increase in mitral valve area of 50% over the baseline or a valve area of at least 1.5 cm2, with no significant increase in mitral regurgitation (=2 grades). Restenosis was defined as a loss of > 50% of the initial gain in mitral valve area. Event free survival is defined as the survival with freedom from NYHA functional class III/IV, re-intervention (BMV/MVR), cardiac death or stroke/TIA. [30, 31]

Preterm was defined as babies born before end of 37 weeks of gestation (less than 259 days) and low birth weight (LBW) was defined as birth weight of less than 2.5 Kg (up to and excluding 2499gm). [40] Malnutrition was graded as per the IAP classification using ICDS growth chart as recommended by

IAP, Stunting was graded as per the Water low's classification, Microcephaly was defined as <2SD from expected. [41, 42]

Follow up:

For analysis, interim follow up was considered as the first follow up after delivery for pregnant group and the 6 months follow up after BMV in control group. Final follow up was taken as the last available follow up in both the pregnant and control group. In case of any re-intervention, the last follow up prior to the re-intervention was considered as the last follow up.

Statistical analysis

Statistical analysis was done using the software SPPS version 11.0. Continuous variables are expressed as mean ± SD, categorical data as percentages. Procedural results were compared using an unpaired Student's t-test. Comparisons between pre procedure, post procedure, interim and last follow-up measurements were performed with a 2-tailed Student's paired *t* test. Categorical variables were compared using Chi square test. Multiple stepwise logistic regression analyses of pre procedure, post procedure and interim data independent variables were performed to determine independent predictors of restenosis and LBW. Event-free survival curves were constructed by the Kaplan-Meier method and survival probabilities were compared by the log-rank test. P value <0.05 was considered as significant.

Observations & Results

Table 1: Baseline data

		Group			P value
		Total	Pregnant BMV group	Control group	
Number of Patients (No.)		184	63	121	
Age (Mean ± SD)		26.6 ± 4.9	26 ± 4.9	26.8 ± 4.9	0.30
Height (Mean ± SD)		151.4±5.6	152.6±6.2	150.2±4.2	0.65
Prior Interventions (Patients)	Total patients {No, (%)}	23 (10.4)	9(12.9)	14(9.1)	0.68
	Single intervention	19 (2.2)	8(1.6)	11 (2.5)	
	Double intervention	4 (2.2)	1(1.6)	3 (2.5)	
Prior Interventions (Interv type)	Total intervention	27 (10.4)	10(12.9)	17 (9.1)	0.60
	CMV	12 (10.4)	5(12.9)	7 (9.1)	
	BMV	15(2.2)	5(1.6)	10 (2.5)	
Rhythm	AF	3 (1.6)	1 (1.6)	2 (1.7)	0.98
Functional class	Class II {No. (%)}	115 (62.8)	33 (53.2)	82 (67.8)	0.054
	Class III	68(37.2)	29 (46.8)	39 (32.2)	
	Class IV	1(0.45)	1(1.6)	0	
CVA/TIA {No. (%)}		3(1.6)	1(1.6)	2(1.65)	0.57
H/o CCF		1(.45)	1(1.6)	0	0.09

Baseline data is illustrated in Table 1. Baseline data were comparable between two groups. Numbers of patients with NYHA functional class III symptoms were slightly more in pregnant group but numbers were not statistically significant. One patient in the pregnant group presented with NYHA class IV symptoms and had to be taken for an emergency BMV.

One patient (1.6%) in pregnant group and 2 (1.7%) in control group were in atrial fibrillation prior to BMV. 1 patient in Pregnant group had prior history of transient ischemic attack (TIA). 2 patients in control group had prior history of cerebro-vascular accident (CVA).

Table 2: Baseline variables in pregnant group

Total study group		62
Previous h/o RF [Count (%)]		10 (16.1)
Diagnosis of RHD	Present pregnancy	34 (54.8)
	Diagnosed previously	28 (45.2)
Previous Pregnancies	Primigravidae [Count (%)]	33 (53.2)
	Single	22 (35.5)
	Two	7 (11.3)
Children	None [Count (%)]	35 (56.5)
	Single	22 (35.5)
	Two	5 (8.1)
Abortions	No [Count (%)]	59 (95.2)
	Yes	3 (4.8)

Baseline data for pregnant group is illustrated in Table 2. History of rheumatic fever was present in 10 (16%) of patients. 34 (54.8%) patients were diagnosed to have rheumatic heart disease during this pregnancy. 33 patients (53.2%) were primigravidae. It was the second pregnancy in 22 patients (35.5%), and third in 7 patients (11.3%). 3 patients (4.8%) had history of prior abortions.

Procedure details in pregnant group are illustrated in Table 3. The mean gestational period at the time of presentation was 21.7 ± 6.9 weeks. The mean gestational period at the time of procedure was 27.9 ± 4.1 weeks. Fluoroscopy time data was available only in 52 patients (83%). The mean fluoro time was 4.5±3.6 minutes with 3 patients having fluoro time >10 minutes

Pre BMV hemodynamic and echo data are illustrated in Table 4. Most of the variables were comparable between the groups. Patients in pregnant BMV group had a higher mean mitral gradient by echo (20.9 ± 5.7 vs 17.7 ± 6.7). Though the mean RVSP was comparable between the groups, the pregnant BMV group had significantly more percentage of patients with severe pulmonary artery hypertension (52.5% vs 37.2%). Incidence of aortic valve disease and tricuspid valve disease was similar between the groups..

Table 3: Procedure details in pregnant group

	Mean ± SD	Median
Gestational age at presentation	21.7 ± 6.9	23.0
Gestational age at BMV	27.9 ± 4.1	28.0
Fluoro time	4.5±3.6	4

Table 4: Hemodynamic and echo data prior to BMV

		Group			p value
		Total	Control	Study	
LA (a) (mm of Hg)		31.7 ± 7.7	31.7 ± 7.4	31.6 ± 8.3	0.876
LA (v)(mm of Hg)		32.8 ± 10.9	32.3 ± 10.7	33.7 ± 11.4	0.446
LA(m)(mm of Hg)		25.3 ± 7.2	24.8 ± 6.8	26.4 ± 7.9	0.143
MVA (cm2) (mean ± SD)		0.8 ± 0.1	0.8 ± 0.2	0.8 ± 0.1	0.639
MS Mean (mm of Hg)		18.8 ± 6.6	17.7 ± 6.7	20.9 ± 5.7	0.002
RVSP(mm of Hg)		60.5 ± 24.3	58.4 ± 24.2	64.4 ± 24.3	0.115
Wilkins score	<8	126(68.3)	83(67.8)	43(69.4)	0.10
	=8	58 (31.7)	39 (32.2)	19(30.6)	
PAH	Mild to mod	74 (40.7)	48 (39.7)	26 (42.7)	0.005
	Severe	79(42.3)	45(37.2)	32 (52.5)	
MR	Mild	89(48.8)	50 (41.3)	39 (63)	0.003
	Moderate	2(0.9)	0	2(3.1)	
AR	Mild to mod	10 (5.5)	5 (4.1)	5 (8.1)	0.261
AS	Mild	8 (4.4)	5 (4.1)	3 (4.8)	0.976
	Moderate	3 (1.6)	2 (1.7)	1 (1.6)	
TR	Severe	38 (20.9)	20 (16.6)	18 (29.0)	0.388

Post BMV hemodynamic and echo data are illustrated in table 5 and outcomes in table 6. Most of the variables were comparable between the groups. Mean MS gradient by echo was significantly more in the pregnant group (7.9 ± 3.2 vs 5.7 ± 2.4). Mean RVSP by echo was slightly higher in pregnant group, but was not statistically significant (43.8 ± 14.7 vs 41.2 ± 14). Moderate mitral regurgitation was seen in 8 patients (12.9%) in pregnant group & 10 (8.7%) in control group and was due to excessive commissural split in all patients. None of the patients required any surgical intervention for mitral regurgitation.

Embolic manifestations in the form of femoral artery thrombosis was seen in 2 patients in each group, one patient in the pregnant group underwent embolectomy under local anesthesia, fully recovered, and had a full-term normal delivery. All others were managed conservatively and subsequently improved. Success was attained in 56 patients (90.3%) in pregnant group & 111 patients (91.7%) in control group and the results were comparable. One patient in the pregnant group, who had to undergo emergency BMV, subsequently died due to sepsis in spite of successful BMV.

Table 5: Hemodynamic and echo data post BMV

		Group			p value
		Total	Control	Study	
LA (a) (mm of Hg)		19.4 ± 5.4	19.8 ± 5.3	18.7 ± 5.5	0.217
LA (v) (mm of Hg)		19.1 ± 7.3	19.2 ± 6.7	18.8 ± 8.2	0.754
LA(m) (mm of Hg)		14.3 ± 4.8	14.5 ± 4.7	14 ± 5	0.461
MVA (cm2) (mean ±SD)		1.6 ± 0.2	1.6 ± 0.2	1.6 ± 0.2	0.769
MS Mean (mm of Hg)		6.5 ± 2.8	5.7 ± 2.4	7.9 ± 3.2	<0.001
RVSP (mm of Hg)		42.1 ± 14.3	41.2 ± 14	43.8 ± 14.7	0.258
MR	=3+ {No. (%)}	18 (9.8)	10 (8.3)	8 (12.9)	0.112
TR grade	Mild to mod	162(87.8)	116 (87.6)	56 (90.3)	0.605
	Severe	13 (7.1)	9 (7.4)	4 (6.5)	
PAH grade	Mild to mod	124(67.8)	82 (67.7)	42 (70)	0.09
	Severe	20 (11)	13 (10.7)	7 (11.7)	

Table 6: Outcomes Post BMV

		Group			p value
		Total	Control	Study	
MR	=3+ {No. (%)}	18 (9.8)	10 (8.7)	8 (12.9)	0.112
MVA (cm2)	<1.5 {No. (%)}	69 (37.7)	46 (38)	23 (37.1)	0.903
	>=1.5	114 (62.3)	75 (62)	39 (62.9)	
MVA change> 50%	No {No. (%)}	17(9.3)	10(8.3)	7(11.3)	0.507
	Yes	166(90.7)	111(91.7)	55 (88.7)	
Successful BMV	No {No. (%)}	16 (8.7)	10 (8.3)	6 (9.7)	0.749
	Yes	167 (91.3)	111 (91.7)	56 (90.3)	
Embolic manifestations		4(2.18)	2(1.65)	2(3.2)	0.08
Mortality		1(0.45)	0	1(1.6)	0.04

Table 7: Pre & post cath data- control group

	Pre BMV		Post BMV		P value
	Mean ± SD	Median	Mean ± SD	Median	
PA(s)(mm of Hg)	53.8 ± 20.5	50.0	40.6 ± 15.8	36.0	<0.001
PA(m)(mm of Hg)	34.7 ± 13.8	30.0	25.2 ± 10.1	23.0	<0.001
TMG(mm of Hg)	17 ± 5.9	17.0	6.7 ± 4.8	5.7	<0.001
MVA (cm2)	0.8 ± 0.2	0.8	1.5 ± 1	1.4	<0.001

Cath data of control group is illustrated in Table 7. Pre and post cath data were comparable to echocardiographic data.

Table 8: Echo data – Interim (6 months) follow up

		Group			p value
		Total (164)	Control (104)	Pregnant (60)	
MVA (cm2) (mean ±SD)		1.6 ± 0.2	1.6 ± 0.2	1.5 ± 0.2	0.249
MS Mean (mm of Hg)		6.7 ± 4.1	6.6 ± 5	6.8 ± 2.1	0.712
RVSP (mm of Hg)		37.1 ± 12.3	38.9 ± 13	33.9 ± 10.2	0.014
MR Grade	Mod {No. (%)}	16 (9.7)	9 (8.7)	7 (11.5)	0.012
AR grade	Mild to mod	10 (6.1)	6 (5.8)	4 (6.6)	0.703
AS grade	Mild	12 (7.6)	7 (7.3)	5 (8.2)	0.525
	Mod	3 (1.6)	2 (1.7)	1 (1.6)	
TR grade	severe	7 (4.3)	6 (5.8)	1 (1.6)	0.175
PAH grade	Mild to mod	97 (63.4)	66 (68.8)	31 (54.4)	0.199
	Severe	7 (4.6)	5 (5.2)	2 (3.5)	

Table 9: Comparison between post BMV and interim (post delivery) data in pregnant group

		Study		
		Mean ± SD	Difference	p
MS mean	Post	7.9 ± 3.2	1.1	0.002
	Interim	6.8 ± 2.1		
RVSP	Post	42.9 ± 14.3	9.2	<0.001
	Interim	33.7 ± 10.2		

Interim follow up echo data are illustrated in Table 8. Mean RVSP was significantly lower in the pregnant group compared to the control group (33.9 ± 10.2 vs 38.9 ± 13). Similarly patients with mild to moderate and severe PAH were comparatively less in pregnant group [31 (54.4%) & 2 (3.5%) vs 66 (68.8%) & 5 (5.2%)], even though it was not statistically significant.

Comparisons between post BMV and interim (post delivery) data in pregnant group are illustrated in table 9. Post delivery there was a significant decrease in echo MS gradients and RVSP (7.9 ± 3.2 vs 6.8 ± 2.1 and 42.9 ± 14.3 vs 33.7 ± 10.2).

Table 10: Echo data- Last follow up

		Group			p value
		Total(182)	Control(120)	Study(62)	
MVA (cm2) (mean ±SD)		1.4 ± 0.3	1.4 ± 0.3	1.4 ± 0.2	0.499
MS Mean (mm of Hg)		7.8 ± 3.5	7.6 ± 3.8	8.2 ± 2.8	0.341
RVSP (mm of Hg)		35.8 ± 10.6	40.3 ± 10.6	36 ± 10.1	0.012
MR Grade	Moderate	19 (10.4)	11 (9.2)	8 (12.9)	0.257
AR grade	Moderate	22 (12.1)	16 (13.3)	6 (9.7)	0.063
AS grade	Mild	18 (9.9)	11 (9.1)	7 (11.3)	0.719
	Moderate	2 (1.1)	2 (1.7)	0 (0)	
TR grade	Mild to mod	162(89)	105 (87.5)	57 (91.9)	0.692
	Severe	12 (6.6)	9 (7.5)	3 (4.8)	
PAH grade	Mild to mod	113 (62.1)	81(67.5)	32 (51.7)	0.026
	Severe	12 (6.6)	9 (7.5)	3(4.8)	

Last follow up echo data is illustrated in Table 10. Mean MS gradient by echo were comparable in both groups (8.2 ± 2.8 vs 7.6 ± 3.8). Mean RVSP was significantly lower in the pregnant group compared to the control group (36 ± 10.1 vs 40.3 ± 10.6). Similarly patients with mild to moderate and severe PAH were significantly less in pregnant group [32 (51.7%) & 3(4.8%) vs 81(67.5%) & 9 (7.5%)].

Table 11: Outcomes at last follow up

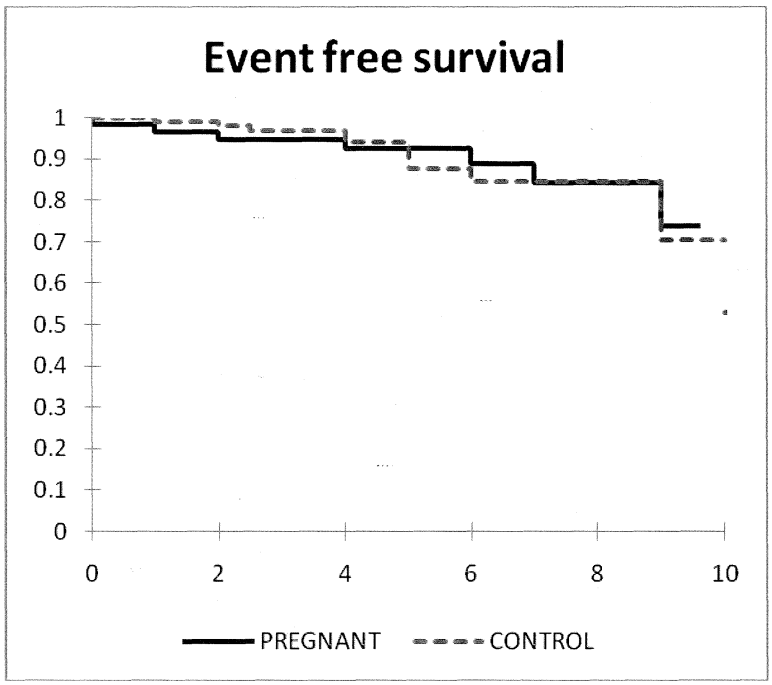
		Group			p value
		Total	Control	Study	
Time to Last Follow up (yrs) (Mean ± SD)		4.51 ± 2.9	4.2 ± 2.7	5.12 ± 2.85	0.215
NYHA FC III-IV {No. (%)}		17 (9.44)	11 (9.2)	6 (9.8)	0.897
Atrial fibrillation		7(3.89)	5(4.2)	2(3.3)	0.20
Re- stenosis		24(13.11)	18(14.87)	6(10)	0.114
Re interventions	Patients {No. (%)}	17 (9.4)	11 (9.2)	6 (9.8)	0.897
	Interventions (No.)	18	11	7	
	Average time (Mean ± SD)	4.8 ± 3.6	5.1 ± 3.9	4.5 ± 3.5	
H/o. SUB BMV	Patients {No. (%)}	12 (6.67)	7 (5.9)	5 (8.2)	0.555
	Interventions (No.)	13	7	6	
	Average time (Mean ± SD)	3.4 ± 1.8	3.8 ± 1.9	2 ± 0	
H/o. SUB MVR	Patients {No. (%)}	5 (2.78)	4 (3.36)	1 (1.6)	0.506
	Interventions (No.)	5	4	1	
	Average time (Mean ± SD)	5 ± 3	5 ± 3.2	4.8 ± 3.1	
Event Free survival		166(90.2)	110 (90.9)	56(88.9)	0.81

Outcomes at last follow up are given in Table 11. Mean time for follow up was 5.12 ± 2.85in pregnant group and 4.2 ± 2.7 in control group and was comparable between groups. NYHA FC II/IV, AF and re-interventions were comparable between groups. Restenosis was seen in 6 patients (9.9%) in pregnant group and 18 patients (14.87 %) in control group.

Even though restenosis was more in the control group, some of the patients continued to be in FC II and as the valve was not suitable for Re-BMV were kept under medical follow up. Re-interventions were done in 6 (9.8%) in pregnant group & in 11 (9.2%) patients in control group, with BMV in [5 (8.2) & 7 (5.9%)] & MVR in [1 (1.6%) & 4 (3.36%)].

Event Free survival

Figure 1: Kaplan-Meier survival curves for both groups



Kaplan-Meier survival curves for both groups are illustrated in figure1. Long-term follow-up event-free survival of both groups was comparable between both the groups. By Log rank test the survival probabilities were no different in both groups (P - 0.9501). Overall event free survival rate was 90 % at 5 years after BMV and 75% at 10 years after BMV

Table 12: Predictors for re-intervention, re-stenosis.

	Variables	Univariate analysis		Multivariate analysis	
		Pregnant BMV	Controls	Pregnant BMV	Controls
Post BMV	MVA	0.01	0.05	0.01	0.02
	LA (m)	0.64	0.02	NS	NS
Change in	MVA	0.05	0.25	NS	NS
Interim FU	MVA	0.02	0.02	0.03	0.04
	RVSP	0.53	0.01	NS	0.01

Predictors for re-interventions and restenosis are illustrated in table 12. On Univariate analysis in the pregnant group post-BMV MVA, change in MVA, Interim (post delivery) MVA were found to be significant. In the control group post-BMV MVA, post-BMV LA mean pressure, interim (6 months follow up) MVA and RVSP were significant. On multivariate analysis only post-BMV MVA, interim follow up MVA were significant predictors for re-intervention in both groups.

Maternal outcomes:

Table 13: Delivery details

Follow up Data	Available	51
Successful delivery	Yes [Count (%)]	48(94.1)
	No	3(5.9)
Timing of delivery	Pre term	3 (6.25)
	Full term	45(93.75)
Delivery Details	Normal [Count (%)]	30 (58.8)
	Elective Cesarean	17 (33.3)
	Emergency cesarean	1 (1.96)
	Still born	1 (1.96)
	MTP/abortion	1 (1.96)
	IUD	1 (1.96)

Delivery details and maternal outcomes are as illustrated in table 13 and 14. Follow up data regarding delivery details was available in 51 patients (82.25%) only. Out of which successful outcomes were seen in 48 (84.1%) patients. One patient had a still birth. Another patient who underwent BMV before 20 weeks of pregnancy subsequently underwent medical termination of pregnancy uneventfully. 1 patient who had a twin pregnancy (prior to BMV only one fetus was viable), had an intrauterine death of the other fetus.

Among the patients with successful outcomes 30 patients (58.8%) had a normal vaginal delivery, 17 (33.3%) underwent elective caesarian and 1(1.96%) had to undergo emergency caesarian (for obstetric reason). 3 patients had a preterm delivery.

On follow up 13 patients (21%) went onto conceive, with normal uneventful deliveries, without any further interventions. One of the patients went through 2 successful pregnancies. Re-interventions were performed in 6 patients (9.7%) due to restenosis and worsening functional class. 1 patient (1.6%) went for MVR and 5 patients (8.1%) had a repeat BMV performed on them. 1 patient required BMV twice during the follow up.

Table 14: Maternal outcomes

Total patients		62
Subsequent number of pregnancies/deliveries	Total [Count (%)]	14 (21.0)
	Single	12 (19.4)
	Twice	1 (1.6)
Subsequent number of interventions	Single [Count (%)]	5 (8.1)
	Twice	1 (1.6)
Subsequent interventions	BMV [Count (%)]	6 (9.7)
	MVR	1 (1.6)
Time for First re BMV (yrs)	1	1 (1.6)
	4	1 (1.6)
	6	1 (1.6)
	7	1 (1.6)

CHILDREN

Table 15: Follow up data of Children (Total 48)

Age (Mean ± SD)		4.8 ± 2.9
Birth weight	Normal {no, (%)}	28 (58.3)
	Low birth weight	19 (39.6)
	Very low birth weight	1 (2.1)
	Average(Mean ± SD)	2.5 ± 0.4
Fetal Complication	None {no, (%)}	45 (93.8)
	Prolonged ICU stay	1 (2.1)
Present Weight	No malnutrition {no, (%)}	33 (68.8)
	Grade 1	9 (18.8)
	Grade 2	6 (12.5)
Present Height	No stunting {no, (%)}	31 (64.6)
	Grade 1	12 (25)
	Grade 2	4 (8.3)
	Grade 3	1 (2.1)
Head circumference	Normal {no, (%)}	44 (91.7)
	Microcephaly	4 (8.3)
	Average(Mean ± SD)	49.8 ± 5.2

Follow up data of children are illustrated in table 15. Complete data was available for 48 children. Mean age was 4.8 ± 2.9 years (range 6 months to 10 years). Mean birth weight was 2.5 ± 0.4. Low birth weight was seen in 19 (39.6%) children and very low birth weight in 1 (2.1%) child.

On follow up for mean duration of 4.8 ± 2.9 years, 33 children (68.8%) had no evidence of any malnutrition, with 9 (18.8 %) & 6 (12.5%) children having grade 1 and grade 2 malnutrition respectively. Microcephaly was noted in 4(8.3%) patients. Grade 1 and grade 2 stunting were noted in 12 (25 %) & 4 (8.3%) children respectively.

All patients had normal mile stones with normal physical and mental development. One child was noted to have mild supravalvular aortic stenosis and peripheral pulmonary stenosis and was kept on medical follow up. No other congenital abnormalities were noted in any other children except one child who had hypospadiasis.

Comparisons between low birth weight and normal birth weight children in regard to their growth are illustrated in table 16. Low birth weight had no significant bearing on malnutrition or stunting on follow up. Microcephaly was significantly common in children with low birth weight, but apparently had no clinical bearing in view of normal development and normal mile stones in all children.

Table 16: Distribution of selected variables according to birth weight

		Birth weight				P value
		Normal		Abnormal		
		Count	Percent	Count	Percent	
Present weight	No malnutrition	21	75.0	12	60.0	0.214
	Malnutrition	7	25.0	8	40.0	
Present Height	No stunting	21	75.0	10	50.0	0.070
	Stunting	7	25.0	10	50.0	
Head circumference	Normal	28	100.0	16	80.0	0.025
	Microcephaly	0	0.0	4	20.0	

Table17: Predictors – low birth weight

	Variable	Univariate analysis	Multivariate analysis
Pre	LA (M)	0.09	NS
Post	MVA	0.01	0.04
	RVSP	0.02	0.04
	LA (M)	0.04	0.03
Change in	MVA	0.05	NS

On Univariate analysis, neither fluoroscopic time nor gestational age had any bearing on low birth weight. Post BMV MVA, post BMV RVSP, post BMV LA mean pressures and change in MVA were significant. On multivariate analysis Post BMV MVA, post BMV RVSP and post BMV LA mean pressures were significant predictors of low birth weight.

Discussion

Baseline clinical data

In the present study history of rheumatic fever was present only in (16%) of patients. 34 (54.8%) of patients had mitral stenosis diagnosed for the first time in the index pregnancy. This outlines the importance of proper screening for heart disease in pregnant patients, especially in developing countries. 33 patients (53.2%) were primigravidae similar to results from Routray et al. Patients in pregnant BMV group had a higher mean mitral gradient by echo and also more percentage of patients had severe pulmonary hypertension. This could be very well explained by the hyper dynamic state of pregnancy which would result in increased mitral gradients and also consequent elevation in left atrial pressures and pulmonary venous and arterial hypertension.

The mean gestational period at the time of procedure was 27.9 ± 4.1 weeks, slightly more than previous studies. The mean fluoro time was 4.5±3 minutes and was comparable to previous studies. Excluding 3 patients with high fluoroscopy times, the mean was only 3.97± 1.5 minutes. Pre procedure mitral valve areas were comparable to most reports from India, which are much smaller than valve areas from the west, probably to differences in body surface area and also indicating the presence of more severe disease in the Indian subcontinent

Immediate results:

In this study immediate success was attained in 90.3% in the pregnant group and 91.7% in the control group. The results were comparable between the groups. The decrease in LA pressure, transmitral gradient and PA pressures was slightly more in the control group, but it was not statistically significant. Mean MS gradient by echo and mean RVSP by echo was slightly higher in pregnant group, but was not statistically significant. This may be due to the hyper dynamic state associated with pregnancy. The results from this study are comparable to results published from other centers. Mitral valve area increased significantly from 0.8 ± 0.1 to 1.6 ± 0.2 cm2 in this series. The final valve area achieved was smaller in this series, probably due to severe disease and presence of severe subvalvular pathology.

Complications:

Apart from femoral artery thrombosis in 2 patients in each group, no other major complications were seen in our study. One patient who presented in pulmonary edema and who had to be taken for emergency BMV, subsequently died after 4 days due to sepsis in spite of a successful BMV. Even though moderate to severe MR (MR 3 to 4+) was seen in 8 (12.9%) in pregnant group and 10 (8.3%) in control group, none required MVR. Thrombo-embolic complications and severe mitral regurgitation were reported in range of 1-2.9% &1.1% to 9.1% respectively in various series.

There is no procedure-related mortality reported in any of the series. 2 occurrences of cardiac tamponade and 1 case of pulmonary thromboembolism, one case of aortic perforation have been reported in earlier studies.

Interim follow up:

Pregnant BMV patients were reassessed post delivery and compared with the control group who were reassessed 6 months after BMV. Mean MVA, transmitral gradients were comparable between groups. Mean RVSP was significantly lower in the pregnant group compared to the control group. Similarly patients with mild to moderate and severe PAH were comparatively less in pregnant group. These results were in contrast to the immediate post BMV data where the mean transmitral gradient and PA pressures were higher in the pregnant group. When the post BM and post delivery results were compared, there was a significant decrease in mean transmitral gradient and PA pressures. This was probably related to the hyperdynamic circulatory state associated with pregnancy.

Final follow up:

Mean follow up was slightly longer in pregnant group (5.12 ± 2.85 vs 4.2 ± 2.7). More than 90% were in functional class I/II in both groups. Final MVA, mean MS gradient was comparable between the groups. Mean RVSP & proportion of patients with severe PAH were lower in pregnant group.

Outcomes at last follow up:

Restenosis was seen in 18(14.87) patients in control group compared to 6 (10%) in pregnant group. Even though the restenosis was more in control group all of them did not go for Reinterventions as most of them were in functional class II, and valves were considered as a very high risk for re-BMV. Re-interventions were comparable to both groups. 90.2% patients in pregnant group and 90.7% patients in control group were in functional class II at end of follow up. 2 patients (3.3%) in pregnant group and 5 patients (4.2%) in control group were in atrial fibrillation at last follow up. Event free survival was comparable in both groups [56 (88.89%) vs 110 (90.9%)].

Delivery details and maternal outcomes:

Of the 63 patients who underwent BMV in the present study, delivery data was available only for 51 patients. Of the 48 who had successfully delivered, 58.8% had normal vaginal delivery. These results were comparable with previous studies. Most of the patients, who underwent cesarean section, underwent the same for obstetric reasons only.

Comparison of maternal outcomes with other studies

Comparisons between the present study and previous studies are illustrated in Table 18 & 19. Compared to other studies the mean gestational age was higher in our study.

55% of patients were diagnosed to have rheumatic heart disease for the first time in the index pregnancy, which was significantly more than previous studies, but similar to the study by Routray et al. Pre procedure valve areas were comparable to most reports from India, which are much smaller than valve areas from the west, indicating the presence of severe disease in the Indian subcontinent. The change in MVA was only 0.8 cm^2, which was less when compared to the western literature and also some Indian studies. This was comparable to the studies by Gupta et al, Algotar et al and studies from Brazil. Similarly the final valve area achieved was smaller in our series, probably due to severe disease and presence of severe subvalvar pathology.

However this did not affect the immediate and long-term outcomes, with the procedural success, complication rates and event free survival not different from other studies. [25, 29, 32-38, 43-45.] Numbers of still births/IUD were also similar to the previous studies. 13 patients on follow up went to have a subsequent ± 1 pregnancy and had a successful outcome. [25, 29, 32-38, 43-45.]

Predictors of re-intervention and restenosis:

Esteves et al in patients undergoing BMV in pregnancy, failed to identify any independent predictor of restenosis. [37] Fawzy et al in their study in non pregnant patients reported event free survival rate of 79% at 10 years and 43% at 15 years in relatively younger patients.

Also event free survival rate was significantly higher for patients with optimal valve anatomy (88% at 10 years and 66% at 15 years). They found that valve anatomy, age and post procedural mitral valve area were predictors of restenosis and event free survival. [46, 47] Similarly by multiple stepwise logistic regressions, post-BMV MVA and interim follow up MVA were identified as independent predictor of re-interventions and event free survival in the present study. In non pregnant group post procedure RVSP was also significant predictor of restenosis. Higher RVSP post procedure might suggest an advanced state of disease, with higher probability of restenosis.

Table 18

Study	Yr	Nos.	Gestational age	Primigravidae(%)	Diagnosis in index preg(%)	Previous intervention (%)	AF (%)	Change in MVA	Procedural success (%)	Major complications (%)	Fluoroscopy	Stillborn/abortion/IUD
Patel et al	1993	19	-	-	-	-	-	-	18(94.7)	1 (5.3)	9.2±3.4	0
Kalra et al	1994	27	-	-	-	-	-	-	26(96.2)	1(3.8)	-	0
Ben farhat et al	1997	44	-	-	-	-	-	-	40(90.9)	4(9.1)	16±1.9	0
Gupta et al	1998	40	21 ±11	27.5	40	-	2.5	0.9	38(95)	2(5)	7.8±1.9	1
Mangione et al	2000	30	24 ± 7	-	-	-	3.3	0.87	28(98.3)	0	19±9	2
Fawzy et al	2001	23	-	-	-	-	-	-	23(100)	0	-	1
Mishra et al	2001	85	22.7±4.1	28.4	35	5.9	23.5	1.25	80(94)	2(2.2)	3.6±3.2	0
Necrolini et al	2002	44	23±6	-	-	11.5	2.3	0.89	42(95)	1(2.3)	-	3
Routray et al	2003	40	24.2±4.6	68	50	-	25	1.08	38(95)	1(2.5)	5.5±3.8	1
Algotar et al	2004	52	-	36	-	-	-	0.9	50(96)	2(4)	4.2±	1
Harikrishnan et al	2005	36	26.5±5.3	55.6	33	0	2.78	0.85	35(97.2)	1(2.8)	5.4 ± 5.8	1
Esteves et al	2006	71	24 ± 7	-	-	7	0	1.1	71(100)	2(2.8)	-	4
Present study	2010	63	27.9±4.1	53.2	54.8	9.1	1.6	0.8	56 (90.3)	2(3.17)	4.5±3	3

Table 19

Study	follow up no.(%)	follow up period	Reinterventions	Fc I-11 on fu	Subsequent pregnancies (%)
Mangione et al	23 (76.7)	5.33±3	2(8.7)	21(91.3)	-
Harikrishnan et al	30(83)	2.68±2	1(3.3)	29(96.7)	4(13.3)
Esteves et al	63(91.5)	3.67±2.1	9(14.3)	53 (84.1)	-
Present study	61(96.8)	5.0±3.3	6 (9.7)	56(90.3)	13(21)

Events free Survival:

Events free survival was 88.89 % in the pregnant group after a mean follow up of 5.12 ± 2.85 years. In the control group the event free survival was 90.9 after a mean follow up period of 4.2 ± 2.7 years. Even though the event free survival was slightly better in control group, it was not significant. Comparatively shorter duration of follow up in the control group could be a reason partly.

Kaplan Meir analysis showed similar survival probabilities between the two groups with a 90 % survival 5 years after BMV, and 75% survival 10 years after BMV in both groups. The results were similar to study by Esteves et al, who in their study compared pregnant patients with age- and gender-matched cohort of 108 patients from the Massachusetts General Hospital BMV database and found comparable survival probabilities. [37.]

Children follow up:

Complete data was available for 48 children. Mean age was 4.8 ± 2.9 years (range 6 months to 10 years). 3 children (6.3%) had a premature delivery, with one requiring prolonged neonatal intensive care. Mean birth weight was 2.5 ± 0.4. Low birth weight was seen in 19 (39.6%) children and very low birth weight in 1 (2.1%) child.

Population studies done from kerala showed average birth weight of 2.8 ±0.40. Sasidharan et al in their study showed that kerala children had a mean birth weight 2.8 ±0.40 kg, which was greater than in Indian contemporaries but consistently <-1 SD below the National Center for Health Statistics reference median throughout childhood. Birth weight significantly predicted body mass (BMI) at 8 years. Socioeconomic status was one of the principal determinants of low birth weight in this study. [48.] Raman kutty et al in another study showed that the average birth weight was 2.84 ± 0.52, and that prematurity, parity and mother's height (<149 cm) were predictors of LBW. [49]

Radhakrishanan et al in another study from our institute showed the prevalence of low birth weight to be around 17.9%. A low maternal socioeconomic status was noted to be the principal determinant of a low birth weight baby. Prevalence of low birth weight in low socioeconomic group was 23% and was statistically significant. Among the low birth weight children 69.8% belonged to low socio economic status.[50]

The prevalence of low birth weight in our study was significantly higher than general population. This could be probably due to the low socioeconomic status (76% of patients belonged to low socio economic status) associated with rheumatic heart disease, the stigmata of chronic inflammatory disease and after effects of low cardiac output associated with severe mitral stenosis.

In our study the average height was 150 ± 4.2, which could have also had an impact on birth weight. Also the mean gestational age at time of pregnancy was slightly more in our study population. Also the children consisted of mixed population from rural Tamilnadu and Kerala. Kamal doss et al also in their study showed that the rate of LBW to be 24.6%, with a mean birth weight of 2.72 ± 0.44 kg in rural Tamilnadu. [51]

On follow up for mean duration of 4.8 ± 2.9 years, the prevalence of malnutrition was 31.2%, with stunting seen in 33.3% of children. This was nearly similar to estimated prevalence of malnutrition and stunting as per the NFHS-3 (table 20). [52] Similar to earlier studies, Low birth weights did not have any significant bearing on malnutrition or stunting on follow up. Microcephaly was significantly common in children with low birth weight, but apparently had no clinical bearing in view of normal development and normal mile stones in all children.

Similar to study by Esteves et al, no correlation was found between gestational age (at the time of PMV) and newborn weight. Similarly neither fluoroscopic time nor pre BMV echo parameters had any bearing on low birth weight. [37] On multivariate analysis Post BMV MVA, post BMV RVSP and post BMV LA mean pressures were significant predictors of low birth weight.

The patients who had a better MVA post BMV, low PA and LA pressures post BMV had less incidence of LBW. This emphasizes the need to achieve a good result PTMC in pregnant patients for better neonatal outcomes.

Table 20:

Table 1 Prevalence of Malnutrition among children (0-59 months) across fifteen major states of India (NFHS-3)

States/Country	*Underweight*			*Stunting*			*Wasting*		
	Rural	Urban	Total	Rural	Urban	Total	Rural	Urban	Total
Haryana	41.3	34.6	39.6	48.1	38.3	45.7	19.7	17.3	19.1
Punjab	26.8	21.4	24.9	37.5	35.1	36.7	9.2	9.2	9.2
Rajasthan	42.5	30.1	39.9	46.3	33.9	43.7	20.3	20.8	20.4
Madhya Pradesh	62.7	51.3	60	51.7	44.3	50	36	31.7	35
Uttar Pradesh	44.1	34.8	42.4	58.4	50.1	56.8	15.2	12.9	14.8
Bihar	57	47.8	55.9	56.5	48.4	55.6	27.4	25.2	27.1
Orissa	42.3	29.7	40.7	46.5	34.9	45.0	20.5	13.4	19.5
West Bengal	42.2	24.7	38.7	48.4	29.3	44.6	17.8	13.5	16.9
Assam	37.7	26.1	36.4	47.8	35.6	46.5	13.6	14.2	13.7
Gujarat	47.9	39.2	44.6	54.8	46.6	51.7	19.9	16.7	18.7
Maharashtra	41.6	30.7	37	49.1	42.3	46.3	18.2	14.1	16.5
Andhra Pradesh	34.8	28	32.5	45.8	36.7	42.7	13	10.7	12.2
Karnataka	41.1	30.7	37.6	47.7	36	43.7	18.2	16.5	17.6
Kerala	26.4	15.4	22.9	25.6	22.2	24.5	18.2	10.9	15.9
Tamilnadu	32.1	27.1	29.8	31.3	30.5	30.9	22.6	21.6	22.2
All 15 states	**44.1**	**32.2**	**41.1**	**49.7**	**39.2**	**47.04**	**19.9**	**16.6**	**19.04**
ALL INDIA	**45.6**	**32.7**	**42.5**	**50.7**	**39.6**	**48**	**20.7**	**16.9**	**19.8**

Source: Kanjilal et al. International Journal for Equity in Health 2010, 9:19 [52]

Comparison of outcomes in children with other studies:

The comparisons are illustrated in table 21. In comparison to the various studies, mean birth weight was lower, but similar to previous study from our institute. Low socioeconomic status with most of the population coming from rural areas might be contributing to this. This could also probably reflect the presence of more severe disease associated with severe sub- valvular pathology in our study. Also the mean height was 150.2±4.2, which might have contributed to LBW as observed in study by Raman kutty et al. [49] Prematurity was seen in 6.3% of patient which was similar to other studies which varied between 8.6-12.6%. Contrary to other studies where neonatal death was seen in 1.4-10%, no death was noted in our study. Most of the procedures were done electively at appropriate time without much maternal and fetal distress & patients were regularly followed up & deliveries managed in tertiary care centers where the services of a cardiologist and a neonatologist/pediatrician were available. Improved level of obstetric & neonatal care might have also contributed to lack of deaths. [29, 34, 36-38, 53]

Table 21

Study	Yr	Nos.	follow up (Yrs) Mean±SD	Birth wt Mean±SD	Preterm No (%)	Prolonged NICU care No (%)	Death No.	Thyroid/malignancy	Growth& development
Mangione et al	2000	23	>5	-	2(8.6)	0	1(4,3)	None	None
Fawzy et al	2001	23	5.1±2.8	-	-	-	2(8.6)	None	None
Kinsara et al	2002	20	5.25±3.25	-	-	-	2(10)	None	None
Routray et al	2003	40	3±1.25	2.9±0.8	-	0	1(2.5)	None	None
Harikrishnan et al	2005	30	2.68±2	2.45±0.5	3(10)	0	1(3.3)	None	None
Esteves et al	2006	71	3.67±2.1	2.8±0.6	9(12.6)	0	1(1.4)	None	None
Present study	2010	51	5.0±3.3	2.5±0.4	3(6.3)	1(2.1)	0	None	None

Conclusion

1. This study emphasizes the importance of proper screening early in pregnancy for detection and treatment of heart disease especially in developing countries.

2. Immediate procedural success, complications, event free survival and restenosis at follow up were comparable between groups..

3. Post-BMV MVA and interim follow up MVA were identified as independent predictor of re-interventions and event free survival.

4. Average mean birth weight was slightly lower in our group of patients with a slightly higher prevalence of low birth weight.

5. LBW had no significant bearing on malnutrition or stunting on follow up

6. On multivariate analysis Post BMV MVA, post BMV RVSP and post BMV LA mean pressures were significant predictors of low birth weight.

7. In the pregnant population, BMV is safe & effective treatment that results in excellent immediate & long-term outcomes for mothers & their offspring.

8. BMV is the procedure of choice to treat symptomatic pregnant women with rheumatic mitral stenosis who are unresponsive to adequate medical treatment.

Bibliography

1. Szekely P, Snaith L. Maternal mortality in rheumatic heart disease. In: Szekely P, Snaith L, editors. Heart desiase and pregnancy. Edinburg: Churchill Livingston; 1974. p 129–133.

2. McFaul PB, Dorman JC, Lamki H, Boyle A. Pregnancy complicated by maternal heart disease: a review of 519 women. Br J Obstet Gynaecol 1988;95:861.

3. Vijaykumar M, Narula J, Aeddy KS, Kaplan EL. Incidence of rheumatic fever and prevalence of rheumatic heart disease in India. Int J Cardiol 1994;43:221 –228.

4. Visser AA, Coetzee EJ, Grolier CJF, Cronje HS. Cardiac disease. In: Cronje HS, Grolier CJF, Visser AA, editors. Obstetrics in Southern Africa. Pretoria: J.L. Van Schaik, 1996. (p. 229).

5. Desai DK, Adanlawo M, Naidoo DP, Moodley J, Kleinschmidt I, Canetti M, Akhter MW. Mitral stenosis in pregnancy: a four year experience at King Edward VIII Hospital, Durban, South Africa. Br J Obstet Gynaecol 2000;107:953 –958.

6. Hameed A, Karaalp IS, Tummala PP, Wani RW, Canetti M, Akhter MW. The effect of valvular heart disease on maternal and fetal outcome of pregnancy. J Am Coll Cardiol 2001;37:83 –99.

7. McAnulty JH, Morton MJ, Ueland K. The heart and pregnancy. Curr Probl Cardiol 1988;13:589–665.

8. Walters WAW, MacGregor WG, Hills N. Cardiac output at rest during pregnancy and puerperium. Clin Sci 1966;30:1.

9. Bragwat AR, Engel PJ. Heart disease and pregnancy. Cardiol Clin 1995;13:163–178

10. Clark SL, Phelan JP, Greenspoon J, Aldahl D, Horeistein J. Labor and delivery in the presence of mitral stenosis: central hemodynamic observations. Am J Obstet Gynecol 1985;152:984–988.

11. Ducey JP, Ellsworth SM. The hemodynamic effects of severe mitral stenosis and pulmonary hypertension during labor and delivery. Int Care Med 1989;15:192–195.

12. Sullivan JM, Ramanathan KB. Management of medical problems in pregnancy: severe cardiac disease. N Engl J Med 1985;313: 304–309.

13. Szekely P, Snaith L. Obstetric care and the fetus in rheumatic heart disease. In: Szekely P, Snaith L, editors. Heari Disease and Pmgnancy. Edinburgh Churchill Livingstone, 1974; 137.

14. Wickman K, Ryden G, Karlberg B. Aplacebo-controlled trial of metopropol in treatment of hypertension in pregnancy. Scad J Clinic Lab Invest 1984; 44 (Supplll69): 90-95.

15. Butters L. Kennedy S, Rubin PC. Atenolol in essential hypertension during pregnancy. BMJ 1990; 301: 587-589.

16. Pavankumar P, Venugopal P, Kaul U, et al. Closed mitral valvotomy during pregancy: a 20-year experience. Scand J Thorac Cardiovasc Surg 1988;22:11–5.

17. Goon MS, Raman S, Sinnathuray TA. Closed mitral valvotomy in pregnancy: a Malayasian experience. Aust N Z J Obstet Gynaecol 1987;27:173–7.

18. Zitnik RS, Brandenburg RO, Sheldon R, Wallace RB. Pregnancy and open-heart surgery. Circulation 969;39(Suppl I):257–262.

19. Becker RM. Intracardiac surgery in pregnant women. Ann Thorac Surg 1983;36:453– 456.

20. Inoue K, Owaki T, Nakamura T, Miyamoto N. Clinical application of intravenous mitral commissurotomy by a new balloon catheter. *J Thorac Cardiovasc Surg* 1984;87:394–402.

21. De Souza JA, Martinez EE Jr, Ambrose JA, Alves CM, , Carvalho AC. Percutaneous balloon mitral valvuloplasty in comparison with open mitral valve commissurotomy for mitral stenosis during pregnancy. *J Am Coll Cardiol* 2001;37:900 –903.

22. Sananes S, Iung B, Vahanian A, Acar J, Salat-Baroux J, Uzan S. Fetal and obstetrical impact of percutaneous mitral commissurotomy during pregnancy. Fetal Diagn Ther 1994;9:218–225.

23. Brent RL. The effect of embryonic and fetal exposure to X-Ray, microwaves and ultrasound; counseling the pregnant and non pregnant patient about these risks. Semin Oncol 1989;16:347– 368.

24. Gray JE. The radiation hazard let's put it in perspective. Mayo Clinic Proc 1979;54:809–813.

25. Ben Farhat M, Gamra H, Betbout F, Maatouk F, Jarrar M, Addad E. Percutaneous balloon mitral commissurotomy during pregnancy. *Heart* 1997;77:564–567.

26. Kalra GS, Arora R, Khan JA, Nigam M, Khalilullah M. Percutaneous mitral valvotomy during pregnancy. *Cathet Cardiovasc Diagn* 1994;33:28 –30.

27. Martinez-Reding J, Cordero A, Kuri J, Martinez-Rios MA, Salazar E. Treatment of severe mitral stenosis with percutaneous balloon valvotomy in pregnant patients. *Clin Cardiol* 1998;21:659–663.

28. Cheng TO. Percutaneous Inoue balloon valvuloplasty is the procedure of choice for symptomatic mitral stenosis in pregnant women. *Cathet Cardiovasc Interventions* 2000;50:413– 417.

29. Fawzy ME, Kinsara AJ, Stefadouros M, Hegazy H, Kattan H, Chaudhary A, Williams E, Al Halees Z. Long-term outcome of mitral balloon valvotomy in pregnant women. *J Heart Valve Dis* 2001;10:153–157.

30. Glazier JJ, Turi ZG. Percutaneous balloon mitral valvotomy. Prog Cardiovasc Dis 1997;40:5–26.

31. Turi ZG, Reyes VP, Raju BS, Raju AR, Kumar DN, Rajagopal P, Srinath K, Peters P. Percutaneous balloon versus surgical closed commissurotomy for mitral stenosis. Aprospective, randomized trial. *Circulation* 1991;83:1179 –1785.

32. Gupta A, Lokhandwala YY, Satoskar PR, Salvi VS. Balloon mitral valvotomy in pregnancy; maternal and fetal outcomes. *J Am Coll Surg* 1998;187:409–415.

33. Nercolini DC, da Rocha Loures Bueno R, Eduardo Guerios E, Tarastchuk JC, Pacheco AL, Pia de Andrade PM, Pereira da Cunha CL, Germiniani H. Percutaneous mitral balloon valvuloplasty in pregnant women with mitral stenosis. *Cathet Cardiovasc Interventions* 2002;57:318 –322.

34. Mangione JA, Lourenco RM, Dos Santos ES, Shigueyuki A, Mauro MF, Cristovao SA, Del Castillo JM, Siqueira EJ, Bayerl DM, Lins Neto OB, Salman AA. Long term follow-up of pregnant women after percutaneous mitral valvuloplasty. *Cathet Cardiovasc Interventions* 2000;50:413– 417.

35. Mishra S, Narang R, Sharma M, Chopra A, Seth S, Ramamurthy S, Prabhakaran D, Goswami KC, Talwar KK, Manchanda SC, Bahl VK. Percutaneous transseptal mitral commissurotomy in pregnant women with critical mitral stenosis. *Indian Heart J* 2001;53:192–196.

36. Routray SN, Mishra TK, Swain S, Patnaik UK, Behera M. Balloon mitral valvuloplasty during pregnancy. *Int J Gynaecol Obstet* 2004;85:18 –23.

37. Esteves CA, Munoz JS, Braga SLN, Andrade J , Sousa JEMR.. Immediate and Long-Term Follow-Up of Percutaneous Balloon Mitral Valvuloplasty in Pregnant Patients With Rheumatic Mitral Stenosis. Am J Cardiol 2006;98:812–816.

38. Harikrishnan S, Anand S, Sivasankaran S, Krishnamoorthy KM et al. Long-Term Outcome of Patients Undergoing Balloon Mitral Valvotomy in Pregnancy. Am J Cardiol 2005;95:1504–1506.

39. Wilkins GT, Weyman AE, Abascal VM. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and the mechanisms of dilatation. *Br Heart J* 1988;60:299 –308.

40. United Nations Children Fund and World Health Organisation, Low birth weight: Country, regional and global estimates. New York: UNICEF; 2004.

41. Nutritional sub-committee of IAP, Report of Convenor, Indian Pediatr 1972; 9: 360.

42. Hetzel BS. Lancet 1983: 2;1126

43. Patel JJ, Mitha AS, Haseen F. Patel N. Naidu R, Chetty S, et al. Percutaneous balloon mitral valvotomy in pregnant patients with tight pliable mitral stenosis. Am Heart J 1993; 125 : 1106-9.

44. Kalra GS, Arora R, Khan JA, Nigam M, Khalillulah M. Percutaneous mitral stenosis during pregnancy. Cathet Cardiovasc Diagn 1994; 33 : 28-30.

45. Krishna Algotar, Atul Nalawade, DG Dhanawat. Balloon Mitral Valvuloplasty : Maternal and Foetal Outcome. Bombay Heart Journal. 2005; 03: 47.

46. Fawzy ME, Shoukri M, Al Buraiki J, et al. Seventeen years' clinical and echocardiographic follow up of mitral balloon valvuloplasty in 520 patients, and predictors of long-term outcome. *J Heart Valve Dis* 2007;16:454–460.

47. Fawzy ME, Shoukri M, El Shaer F, Osman A, Ibrahim IE, Memon M, Shah S. 18 years follow-up results of mitral balloon valvuloplasty in 531 consecutive patients and predictors of long-term outcome. Heart Views 2007;8:130-41

48. Brown N, Sasidharan CK, Fisher D. Early growth and markers of cardiovascular risk in Keralan children in the Integrated Child Development Scheme. Public Health Nutr. 2010 Jul;13(7):1042-8. Epub 2009 Sep 22.

49. Raman Kutty V. Why low birth weight is still a problem. Discussion paper No.57, Kerala Research Programme on local level development centre for developmental studies Thiruvananthepuram. ISBN No. 81-87621-60-5.

50. T. Radhakrishnan, K.R. Thankappan, R.S. Vasan, P.S. Sarma. Socioeconomic and Demographic Factors Associated with Birth Weight: A Community Based Study in Kerala. Indian Pediatrics 2000;37: 872-87.

51. Theodore Kamaladoss, R. Abel and V. Sampathkumar. Epidemiological co-relates of low birth weight in rural TamilNadu. Indian Journal of Pediatrics Volume 59, Number 3, 299-304.

52. Kanjilal et al. Nutritional status of children in India: household socio-economic condition as the contextual determinant International Journal for Equity in Health 2010, 9:19.

53. Kinsara AJ, Ismail O, Fawzi ME. Effect of balloon mitral valvoplasty during pregnancy on childhood development. Cardiology 2002;97:155 –158.

GLOSSARY

AF	- Atrial fibrillation
BMV	- Balloon mitral valvuloplasty
BSA	- Body surface area
LA	- Left atrium
LBW	-Low Birth Weight
LV	- Left Ventricle
MR	- Mitral regurgitation
MS	-Mitral regurgitation
MVA	- Mitral valve area
MVR	-Mitral valve replacement
NS	-Non significant
NYHA	-New York Health Association
PAH	-Pulmonary arterial hypertension
PASP	-Pulmonary artery systolic pressures
RVSP	- Right ventricular systolic pressures
TEE	-Trans-esophageal echocardiography
TTE	- Trans-thoracic echocardiography
TIA	-Transient ischemic attack
TMG	-Trans-mitral gradient
2D	-Two dimensional

Master Chart

CONTROL POPULATION- DEMOGRAPHICS

PRE PROCEDURE

Hospital No	name	Age	Date of procedure	Functional class	Prior interventions((number)	H/o. Prior CMV((number)	H/o. Prior OMV(number)	H/o. Prior BMV(number)	H/O CVA(0-NO, 1-YES)	Rhythm (0-SR, 1-AF)	LA(SIZE)	MVA (Avg)	MS Mean	MR(Grade)	AR(Grade	AS(Grade)	TR(Grade)	RVSP(mm of Hg)	LA (a) (mm of Hg)	LA (v)(mm of Hg)	LA(m)(mm of Hg)	PA(m)(mm of Hg)	TMG(mm of Hg)
10.1.07	Hancya.A.A	28	20.10.03	3	0	0	0	0	0	0	31	0.85	30	2	2	2	1	45	42	30	24	34	24
210204	Prabha.V	20	29.3.03	3	0	0	0	0	0	0	46	0.85	21	1	0	0	1	30	38	42	33	30	17
211835	Murugesari	23	20.2.03	2	0	0	0	0	0	0	37	1.1	17	1	0	0	1	38	30	40	25	18	19.4
216872	Thiru selvi	22	5.7.03	2	0	0	0	0	0	0	41	1.1	19	2	2	0	2	50	32	30	22	28	21.4
218259	Deepika	26	19.9.03	3	0	0	0	0	0	0	58	0.7	15	2	2	0	3	70	28	21	19	51	12.3
216088	Suma.A.	24	31.07.03	2	0	0	0	0	0	0	42	0.9	14	2	0	0	2	65	40	48	38	47	24.3
220800	Mnju .S	23	19.01.04	3	0	0	0	0	0	0	49	0.7	22	0	0	0	2	58	36	36	26	35	19.6
218653	Sakkala K	32	13.02.04	2	0	0	0	0	0	0	48	0.8	8	2	2	2	2	55	35	35	32	36	NA
208310	Selvi	30	12.10.02	2	0	0	0	0	0	0	40	0.83	30	1	0	0	3	77	32	36	22	30	10
208390	Anie	33	24.10.02	3	0	0	0	0	0	0	45	1.15	25	2	0	0	2	93	40	45	35	65	25
208238	Seenath	30	5.10.02	2	0	0	0	0	0	0	46	0.95	31	2	3	0	2	67	40	36	27	40	18.3
211018	Marammal	26	30.12.02	2	0	0	0	0	0	0	41	0.9	25	1	0	0	2	55	28	20	20	24	13.5
194914	Elsy Binny	30	24.07.01	2	0	0	0	0	0	0	47	0.85	9	2	0	0	2	43	36	46	26	35	17.7
207183	Haleema K	21	18.10.02	2	0	0	0	0	1	0	49	0.8	23	1	0	0	1	40	46	38	32	35	25
204474	Mini Harida	30	22.06.02	2	0	0	0	0	0	0	45	1.07	17	1	1	0	2	52	38	36	25	28	13
204732	Sudha A	20	26.6.02	3	0	0	0	0	0	0	36	0.98	16	2	0	0	2	44	30	25	20	24	13.6
204868	Rajalakshm	19	23.10.02	2	0	0	0	0	0	0	34	1.05	12	2	0	0	2	25	22	24	18	18	15
202664	Radhika	21	25.4.02	2	0	0	0	0	0	0	42	0.7	16	0	0	0	2	46	13	28	22	24	8
202665	Rasheeda	25	25.4.02	3	0	0	0	0	0	0	45	0.6	30	2	2	0	2	36	35	45	31	42	22.8
203053	Chithayee I	24	29.04.02	2	0	0	0	0	0	0	43	0.89	24	1	2	0	3	100	36	38	28	40	17.8

203751	Shailaja	25	14.5.02	3	0	0	0	0	0	0	44	1	27	1	3	1	3	87	40	50	30	43	23
200135	Jaisy K	25	28.8.02	2	0	0	0	0	0	0	34	1	15	2	3	0	2	46	35	36	24	26	10.3
200867	Alma	23	15.2.02	3	0	0	0	0	0	0	44	0.9	10	1	2	0	2	45	32	38	22	25	NA
199756	Sudha C	21	24.05.02	2	0	0	0	0	0	0	44	0.8	18	0	3	1	3	52	37	40	32	38	9.6
195466	Radha B	32	8.9.01	2	0	0	0	0	0	0	35	0.78	8	2	0	0	2	30	24	20	22	24	15.8
196895	Amutha	29	16.2.04	2	0	0	0	0	0	0	45	0.75	28	0	2	0	2	89	40	40	33	43	25
9802410	Usha G	23	3.4.98	2	0	0	0	0	0	0	45	0.85	9	0	2	0	2	44	20	21	16	19	11
9802740	Bijiamma G	22	4.4.98	2	0	0	0	0	0	0	44	0.8	9	1	0	0	NA	NA	NA	NA	24	24	11.5
9801645	Devu P.K.	20	7.7.98	2	0	0	0	0	0	0	34	0.95	14	0	3	0	1	40	NA	NA	18	26	7.4
9804780	Anitha	18	29.10.98	2	0	0	0	0	0	0	40	0.9	20	0	0	0	2	45	42	34	27	31	23.5
9806988	Rajesswari	26	10.9.98	3	0	0	0	0	0	0	34	0.75	10	0	0	0	2	100	40	35	28	78	27
9710517	Honey	26	1.6.98	2	0	0	0	0	0	0	42	0.85	16	1	0	0	2	58	34	38	27	32	17
225063	Ummukulus	32	23.4.04	3	1	0	1	0	0	0	54	0.5	33	0	0	0	3	80	28	32	24	50	16.85
225065	Latha	25	30.10.04	3	0	0	0	0	0	0	53	0.95	17	1	0	0	2	67	33	26	22	34	15
225084	Shahinsha	23	20.3.04	2	0	0	0	0	0	0	32	0.7	27	0	0	0	2	130	34	34	29	52	25
226541	Somy sunn	26	20.4.04	2	0	0	0	0	0	0	46	1.05	10	1	0	0	1	38	28	30	20	25	16
226550	Jebarlin	20	17.5.04	2	0	0	0	0	0	0	34	0.85	15	1	2	0	2	66	34	40	30	45	26.8
9002675	Ponnamma	35	22.9.01	2	0	0	0	0	0	0	32	0.85	12	1	0	0	2	51	22	19	20	22	10
9003504	Safeera	24	12.8.02	2	2	1	0	1	0	0	53	1.05	21	2	2	0	3	75	45	65	44	42	25
9504746	Hema	23	10.7.00	2	1	0	0	1	0	0	46	0.85	18	0	0	0	3	60	40	20	20	27	14.93
9804753	Kunjamma	37	20.6.01	3	0	0	0	0	0	0	42	0.85	10	0	0	0	2	73	32	25	23	65	9
9507629	Subaida M	30	24.5.00	2	0	0	0	0	0	0	53	0.85	19	2	2	0	2	105	45	46	38	68	24.5
9601229	Shiny	21	12.2.00	2	0	0	0	0	0	0	46	0.75	12	1	2	0	2	38	20	23	18	14	9
9508601	Sjithra	42	21.12.01	3	0	0	0	0	0	0	47	0.65	30	2	0	0	4	90	36	42	32	45	22
9503869	Lissy KM Z	28	26.9.00	2	1	0	0	1	0	0	NA	0.7	16	0	2	0	2	59	36	32	25	30	17.3
9700839	Aysha Bai	31	19.01.00	3	0	0	0	0	0	0	42	0.85	12	2	0	0	2	38	14	16	11	16	8
9701664	Simi L	20	20.4.01	2	0	0	0	0	0	0	41	1	12	1	1	0	2	47	32	25	21	35	12.9
9702172	Christy	38	10.8.01	3	0	0	0	0	0	0	41	0.95	26	1	1	0	3	110	36	37	28	57	12.5
9605680	Sindhu KP	26	9.5.02	2	0	0	0	0	0	0	34	0.75	8	1	0	0	2	25	20	18	13	19	8.5
9905081	Sheela	24	24.2.01	3	0	0	0	0	0	0	42	0.45	24	0	0	0	2	90	30	50	30	56	28
9906050	Geetha	24	28.01.00	2	0	0	0	0	0	0	45	1	13	2	0	0	2	48	22	24	20	25	9.1
9608745	Tresa L	37	23.3.02	3	0	0	0	0	0	0	38	1.1	6	1	2	0	2	47	26	22	16	13	14
227932	Usha S	19	21.7.04	2	0	0	0	0	0	0	48	0.57	17	2	0	0	3	93	36	42	33	60	25.7
230539	Sheeja A	22	29.11.04	2	0	0	0	0	0	0	38	0.9	22	1	0	0	2	72	36	33	21	64	10
223362	Soudha K	32	28.01.07	3	0	0	0	0	0	0	35	0.92	19	1	0	0	1	40	28	26	20	25	17.5
208356	Aneesha	25	4.7.06	3	0	0	0	0	0	0	52	0.42	21	2	0	0	3	110	37	50	35	64	21
212351	Selva sund	32	10.1.07	2	1	0	0	1	0	0	42	0.9	12	0	0	0	1	30	28	38	26	28	11
180351	Anitha k	29	11.8.05	3	0	0	0	0	0	0	40	0.55	15	2	0	0	3	60	36	30	26	44	18

244204	Esakkiamm	27	12.1.05	2	0	0	0	0	0	0	36	0.7	19	2	2	0	2	50	22	24	18	21	12
245993	Naseema b	37	4.1.06	2	0	0	0	0	0	0	39	0.7	27	1	0	0	2	60	28	28	24	45	15.4
243710	USHA K	30	18.1.06	2	0	0	0	0	0	0	34	0.9	24	2	2	0	2	95	40	42	24	50	19.5
242636	Balamani	24	17.7.06	2	0	0	0	0	0	0	32	0.9	11	1	2	0	1	30	34	40	31	26	23.67
201255	Rajani	32	22.8.05	3	1	0	0	1	0	0	46	0.7	18	1	0	0	3	70	28	28	22	20	13.5
236507	Sumabenn	25	8.6.05	2	0	0	0	0	0	0	32	0.85	11	0	2	0	2	40	36	26	24	24	12
236560	Mallika	27	3.10.06	3	0	0	0	0	0	0	35	0.8	10	1	2	0	2	100	36	48	28	45	25.17
237896	Jeyanthi	32	2.4.05	3	0	0	0	0	0	0	37	0.85	34	1	0	0	2	80	36	42	34	41	23.2
239691	Leena V	31	8.6.05	2	0	0	0	0	0	0	38	0.75	10.5	2	2	0	2	45	20	17	15	26	10.5
245741	Esakkiamm	25	17.10.05	2	0	0	0	0	0	0	38	0.8	32	1	0	0	2	60	32	38	27	44	21.3
246491	Amaravathi	32	11.1.06	3	0	0	0	0	0	0	31	0.55	28	2	0	0	2	130	40	55	36	62	20
245399	Selvakuma	26	27.10.05	2	0	0	0	0	0	0	40	0.5	15	2	0	0	2	80	32	26	26	32	15.56
278178	Sathyabha	31	09.10.07	3	0	0	0	0	0	0	40	0.85	21	2	0	0	0	NA	NA	NA	18	26	13
277558	Solly john	32	08.08.08	2	0	0	0	0	0	0	49	0.85	26	1	0	0	3	70	26	33	30	35	23
275703	Joseph ma	25	15.04.08	3	0	0	0	0	0	0	37	0.5	12	2	0	0	2	58	22	24	22	28	16.7
270633	Sheeba	27	21.09.07	3	0	0	0	0	0	0	29	0.8	19	1	2	0	2	60	32	28	27	44	18.5
269572	Manju T	27	22.07.09	2	0	0	0	0	0	0	38	0.8	16	2	2	0	2	NA	19	18	16	18	NA
260599	Shakeela	29	03.07.07	3	0	0	0	0	0	0	40	0.93	11	0	0	0	1	25	28	18	18	25	21.39
259662	Mini kunjun	27	27.03.07	2	0	0	0	0	0	0	54	0.87	18	0	2	0	2	50	40	54	40	28	19.25
9906939	Shamia be	29	30.08.07	2	0	0	0	0	0	0	50	0.84	25	2	2	0	2	85	38	36	30	26	17
9501272	Prema S	28	11.08.06	2	2	1	0	1	0	0	70	1.05	20	2	0	0	2	50	40	44	36	30	20.8
9204083	Uma C	26	28.09.05	2	1	1	0	0	0	0	35	0.92	22	2	0	0	2	50	30	31	26	18	13.7
301829	Sheema	26	05.10.09	2	0	0	0	0	0	0	41	0.95	10	1	0	0	2	27	25	38	26	28	12.5
300071	Jyothi laxm	38	26.09.09	2	0	0	0	0	0	0	36	0.97	16	2	0	0	3	120	34	28	26	42	NA
279415	Sakunthala	26	17.09.08	2	0	0	0	0	0	0	45	0.7	12	2	2	0	2	30	28	28	22	50	NA
8802766	Chandri KC	31	27.02.07	2	1	1	0	0	0	0	52	0.68	10	2	0	0	1	30	40	46	26	32	21.2
258442	Sheeja tito	27	10.09.07	2	0	0	0	0	0	0	30	0.95	16	2	0	0	2	60	22	14	12	24	8
258246	Maya	29	06.03.07	2	0	0	0	0	0	0	44	1.05	13	2	0	0	1	30	32	22	26	25	10
255464	Kadeeeja n	30	04.08.06	2	0	0	0	0	0	0	47	0.6	10	1	0	0	1	55	40	54	36	45	27.5
253962	Pushpalath	33	15.09.06	2	0	0	0	0	0	0	44	0.7	17	1	2	0	3	70	46	52	36	47	24.55
253602	Shijina	22	08.08.06	2	0	0	0	0	0	0	47	0.95	15	2	2	1	2	45	29	23	21	30	17.14
253454	Mariamma	20	10.07.05	2	0	0	0	0	0	0	43	0.6	22	1	0	0	2	68	38	55	38	42	28.75
252691	Sathya G	24	27.07.06	2	0	0	0	0	0	0	44	0.95	29	2	0	0	2	70	27	27	21	34	13.22
252419	Sarala P	32	27.05.06	2	0	0	0	0	0	0	40	0.63	27	2	0	0	2	40	34	44	24	45	25.2

250687	Maya V	20	17.03.06	2	0	0	0	0	0	0	42	0.94	17	2	0	0	2	65	41	26	27	30	18.66
247291	Mini johnso	29	05.02.05	2	0	0	0	0	0	0	46	0.82	22	2	2	0	2	45	28	28	23	25	16.36
246534	Girija	27	05.07.06	2	0	0	0	0	0	0	30	0.9	13	1	2	0	1	25	18	12	12	13	13.7
262157	Smitha Vin	27	19.02.07	2	0	0	0	0	0	0	40	0.88	21	2	0	0	2	65	36	40	26	25	19.41
274247	Shanmuga	25	10.07.09	2	0	0	0	0	0	0	56	0.65	14	1	2	0	2	50	39	42	32	42	24
258883	Kalaivani N	29	04.11.06	2	0	0	0	0	0	0	49	0.9	29	1	2	1	3	55	40	30	29	26	18
238742	Geetha A	21	07.05.08	3	1	0	0	1	0	0	43	0.7	26	2	0	0	2	70	34	38	29	50	24
9504914	Nisha ph	22	06.02.08	2	2	0	0	2	0	0	46	0.8	13	1	0	0	2	35	na	NA	19	na	na
273428	Mumtaz	29	16.01.08	2	0	0	0	0	0	0	33	0.7	14	2	0	0	1	30	25	21	21	27	13
9403894	Lakshmi	27	14.01.08	2	1	1	0	0	0	0	38	0.85	10	1	0	0	1	40	30	24	22	27	16
265171	Chellam	33	16.11.09	3	0	0	0	0	0	0	50	0.85	19	2	2	0	2	35	28	26	16	22	na
297320	Mini G	21	20.12.09	2	0	0	0	0	0	0	31	0.7	24	1	0	0	2	55	18	16	14	20	12
294658	Syed Rabiy	38	09.10.09	3	0	0	0	0	0	0	43	0.6	25	2	0	0	3	65	30	36	30	40	17
292809	Thangam F	18	30.09.09	3	0	0	0	0	0	0	39	0.6	24	1	0	0	2	100	40	38	36	50	24
291254	Rajani C	20	10.11.09	3	0	0	0	0	0	0	32	0.9	25	1	0	0	2	85	20	16	13	50	13
228536	Selvalxmi	25	25.03.09	2	1	0	0	1	0	0	34	0.95	11	2	0	0	2	35	29	25	20	24	25.5
289275	Sarithamol	29	28.02.09	2	0	0	0	0	0	0	43	1.3	11	1	2	1	2	30	23	19	18	22	18
9005394	Shibi pappu	32	07.12.09	2	1	1	0	0	0	0	41	1.1	19	2	0	0	2	22	26	20	14	15	12
289442	Mary pushp	25	27.07.09	2	0	0	0	0	0	0	32	0.83	9	1	0	0	1	20	32	28	22	23	9
289981	josina joby	26	18.03.09	2	0	0	0	0	0	0	35	0.75	20	2	0	0	2	75	35	30	31	na	na
290051	Packiyalaxı	24	18.02.09	3	0	0	0	0	0	0	41	0.65	16	1	0	0	3	100	32	27	25	54	20
8800289	Sareena M	30	21.8.95	2	0	0	0	0	0	1	51	0.9	14	0	0	0	2	43		33	23	35	12.5
9503970	Jameela	25	19.7.95	3	0	0	0	0	0	0	41	0.95	13	1	0	0	1	47	20	15	14	16	10
9505315	Ramadevi	22	26.10.95	2	0	0	0	0	0	0	41	0.85	10	1	0	0	2	55	29	26	20	30	2.3
9506575	Pechiamma	23	22.01.96	3	0	0	0	0	0	0	43	0.85	18	0	0	0	1	46	24	29	20	40	14
9507200	Leelamma	28	6.02.96	3	0	0	0	0	0	0	38	0.8	10	0	0	0	2	40	18	19	12	16	na
9503062	Bindhu	21	7.7.95	2	0	0	0	0	1	0	52	1.1	15	0	0	0	2	42	30	26	20	40	11
9504423	Vanaja	32	14.9.95	3	0	0	0	0	0	0	42	1.08	8	0	0	0	2	55	25	25	20	27	9.5
9507950	Lissy	30	29.4.98	2	0	0	0	0	0	1	55	0.85	12	1	0	0	2	60	43	42	32	65	22.6

			POST PROCEDURE																						
name	MVA(Cath)	PAH grade(0-none, 1-mild, 2- moderate, 3- seve	POST MVA (Avg)	MR	MR > 3+ (Y/N)	Commissural tear	MVA Average (1- < 1.5; 2->= 1.5)	MVA CHANGE > 50%	FAIR RESULT (0- NO, 1-YES)	MS Mean (mm of Hg)	AR(grade)	AS(Grade	TR(Grade)	RVSP	LA (a)	LA (v)	LA(m)	PA(s)	PA(m)	TMG	MVA(Cath)	PAH grade(0-none, 1-mild, 2- moderate, 3- seve	Functional class	Rhythm(0-sr, 1-af)	MVA (Avg)
Hancya.A.A	0.9	2	1.45	2	0	0	1	1	1	10	2	2	2	44	22	16	12		18	18	1.7	0	1	0	1.5
Prabha.V	0.7	1	1.9	2	0	0	2	1	1	3	0	0	1	30	27	25	18	32	24	5.2	1.6	0	1	0	2
Murugesari.B	0.67	0	1.45	2	0	0	1	0	0	6	0	0	1	38	20	18	12	25	17	8	1	0	1	0	1.6
Thiru selvi	0.34	1	1.75	2	0	0	2	1	1	10	3	0	2	50	22	20	16	40	25	9.8	0.7	1	1	0	1.7
Deepika	0.84	3	1.55	2	0	0	2	1	1	6	2	0	2	49	20	14	14	48	32	5.1	1.2	2	1	0	1.5
Suma.A.	0.65	3	1.48	2	0	0	1	1	1	3	0	0	2	48	26	24	21	46	34	7.5	1.5	2	1	0	1.6
Mnju .S	0.61	2	1.4	2	0	0	1	1	1	9	0	0	2	48	24	14	12	36	24	6.7	1.6	0	1	0	1.6
Sakkala K	NA	2	1.6	2	0	0	2	1	1	4	2	3	2	42	30	30	22	38	26	NA	NA	1	NA	NA	NA
Selvi	1.14	2	2	2	0	0	2	1	1	7	0	0	2	56	26	34	18	32	32	3	2.2	2	1	0	1.9
Anie	0.8	3	2.15	3	1	1	2	1	1	11	0	0	3	69	20	24	20	70	42	9	1.8	3	2	0	2
Seenath	0.73	3	1.4	2	0	0	1	0	0	4	3	0	2	50	28	20	16	40	30	12	1	2	2	0	1.5
Marammal	0.86	0	1.5	1	0	0	2	1	1	10	0	0	1	45	20	20	12	40	20	4.4	1.4	0	1	0	1.6
Elsy Binny	0.8	2	1.9	2	0	0	2	1	1	3	0	0	2	36	23	18	13	34	20	4.2	1.8	0	1	0	2.1
Haleema K	0.52	2	1.5	2	0	0	2	1	1	4	0	0	1	38	20	16	14	40	26	5	1.4	1	1	0	1.7
Mini Haridas	0.8	1	1.7	1	0	0	2	1	1	3	1	0	2	38	30	22	16	38	20	5.7	1.3	0	1	0	1.9
Sudha A	0.75	0	1.56	2	0	0	2	1	1	7	0	0	2	30	18	13	11	24	14	3.6	1.3	0	1	0	1.5
Rajalakshmi	0.8	0	2	2	0	0	2	1	1	6	0	0	2	20	8	8	7	20	12	4	1.7	0	1	0	2.1
Radhika	1.4	0	1.55	2	0	0	2	1	1	4	0	0	2	36	22	20	13	32	24	5	2.1	0	1	0	1.5
Rasheeda	0.6	3	1.75	2	0	0	2	1	1	4	2	0	2	46	20	18	13	35	22	4.2	1.7	0	1	0	1.9
Chithayee M	0.42	3	2	1	0	0	2	1	1	2	2	0	2	44	16	14	8	44	20	43	1.6	0	1	0	1.7

Shailaja	0.9	3	1.95	2	0	0	2	1	1	4	3	1	2	41	28	35	23	na	na	11	1.6	1	1	0	1.8
Jaisy K	0.86	1	1.5	2	0	0	2	1	1	6	2	0	2	40	24	23	14	30	20	6.8	1.2	0	1	0	1.4
Alma	NA	1	1.52	1	0	0	2	1	1	11	2	0	2	38	22	26	18	26	12	NA	NA	0	1	0	1.4
Sudha C	0.86	2	2.2	2	0	0	2	1	1	2	3	1	2	40	26	28	22	40	23	5.2	1.3	0	1	0	2
Radha B	0.58	0	1.75	2	0	0	2	1	1	4	0	0	2	30	16	18	12	26	18	5	1.5	0	1	0	1.5
Amutha	0.63	3	2.05	1	0	0	2	1	1	7	2	0	2	47	26	26	20	55	38	8	1.4	2	1	0	1.9
Usha G	0.9	0	1.45	1	0	0	1	1	1	5	2	0	1	20	14	16	7	26	14	3	2.4	0	1	0	1.7
Bijiamma G	1.56	0	1.29	1	0	0	1	1	1	8	0	0	0	NA	NA	NA	17	32	21	3	1.7	0	2	0	1.2
Devu P.K.	0.95	1	1.7	1	0	0	2	1	1	4	0	0	1	36	NA	NA	14	28	18	4.4	1.8	0	1	0	1.8
Anitha	0.8	2	1.9	1	0	0	2	1	1	3	0	0	1	40	18	13	11	38	24	5	2	0	1	0	2
Rajesswari	0.5	3	1.27	1	0	0	1	1	1	6	0	0	2	64	20	16	12	65	39	4	1.2	2	1	0	1.3
Honey	0.86	2	1.94	1	0	0	2	1	1	4	0	0	2	48	32	24	16	35	25	5.6	1.4	1	1	0	1.7
Ummukulusu	0.74	4	1.65	2	0	0	2	1	1	11	0	0	3	64	12	18	12	75	46	5.7	1.3	3	2	0	1.7
Latha	0.52	2	1.75	2	0	0	2	1	1	5	0	0	2	50	20	12	11	28	18	2.1	1.6	0	2	0	1.8
Shahinsha Mol	0.59	4	1.73	1	0	0	2	1	1	6	0	0	2	74	8	14	13	50	35	7.5	1.2	2	2	0	1.5
Somy sunny	1.04	1	1.6	1	0	0	2	1	1	8	0	0	0		16	20	16	30	18	8	1.6	0		0	1.5
Jebarlin	0.78	3	1.45	1	0	0	1	1	1	8	2	0	0		20	20	12	40	26	9.4	1.4	1	1	0	1.6
Ponnammai S	1	0	1.9	0	0	0	2	1	1	4	0	0	2	30	20	16	12	24	16	4.3	1.2	0	2	0	1.9
Safeera	0.5	3	1.3	2	0	0	1	0	0	12	2	0	2	70	20	22	18	45	30	6.2	11	2	2	0	1.3
Hema	1.01	1	1.6	1	0	0	2	1	1	7	0	0	3	50	22	13	13	38	23	8.4	1.3	0	2	0	1.5
Kunjamma Varg	0.8	3	1.8	0	0	0	2	1	1	6	0	0	2	64	18	14	12	90	55	2.8	1.2	3	2	0	1.7
Subaida Moidu	0.49	3	1.25	2	0	0	1	1	1	6	2	0	2	70	28	30	24	78	45	14	1.1	3	2	0	1.6
Shiny	0.4	0	1.5	2	0	0	2	1	1	6	2	0	2	36	20	20	15	18	10	1.4	1.3	0	2	0	1.5
Sjithra	0.4	3	1.73	2	0	0	2	1	1	5	0	0	4	68	22	25	19	73	33	7.2	0.7	2	2	0	1.9
Lissy KM Zacha	0.7	2	1.55	1	0	0	2	1	1	7	2	0	2	50	20	19	16	32	20	5.5	1.4	0	2	0	1.5
Aysha Bai	1.1	0	1.65	2	0	0	2	1	1	6	0	0	2	38	10	12	6	24	16	3	2	0	2	0	1.5
Simi L	0.7	2	1.95	2	0	0	2	1	1	6	1	0	2	40	21	18	14	32	14	5.8	1.3	0	1	0	1.9
Christy	0.49	3	1.85	2	0	0	2	1	1	6	1	0	0		30	30	20	55	45	7.1	0.9	3	2	0	1.8
Sindhu KP	1.15	0	1.85	2	0	0	2	1	1	3	0	0	2	30	12	10	8	26	16	4.2	2	0	2	0	1.9
Sheela	0.4	3	1.35	3	0	0	1	1	1	10	0	0	2	80	22	41	21	90	40	5.4	1.1	3	1	0	1.5
Geetha	1	1	1.5	2	0	0	2	1	1	5	0	0	2	45	14	10	10	24	14	3.8	1.5	0	1	0	1.7
Tresa L	0.8	0	1.65	2	0	0	2	1	1	6	2	0	2	48	24	21	24	30	22	8.9	1.2	0	2	0	1.6
Usha S	0.4	3	1.15	3	1	1	1	1	1	13	0	0	2	60	24	40	30	80	56	14	0.6	3	2	0	1.3
Sheeja A	0.6	3	1.55	2	0	0	2	1	1	6	0	0	0		13	14	11	45	32	4	1.3	2	1	0	1.9
Soudha K	1.07	1	1.4	1	0	0	1	1	1	5	0	0	1	30	14	14	8	28	18	4	2	0	1	0	1.7
Aneesha	0.35	3	0.9	3	1	1	1	1	1	8	0	0	3	90	23	21	21	90	50	8	1.3	3	1	0	1.2
Selva sundari	1.1	1	1.55	0	0	0	2	1	1	3	0	0	1	35	20	24	17	40	26	3.3	2.2	1	2	0	1.4
Anitha k	0.8	3	1.45	2	0	0	1	1	1	6	0	0	3	60	16	10	8	38	25	4	1.6	1	2	0	1.4

Esakkiammal s	0.74	0	1.35	3	1	1	1	1	1	5	2	0	2	25	12	14	9	24	12	6	1.2	0	1	0	1.5
Naseema beevi	0.9	3	1.25	1	0	0	1	1	1	5	0	0	2	35	18	16	15	55	40	8.6	1.3	3	1	0	1.2
USHA K	0.43	3	1.5	2	0	0	2	1	1	4	2	0	2	65	21	17	13	48	35	6	1.1	2	1	0	1.6
Balamani	0.81	1	1.24	2	0	0	1	0	0	4	2	0	1	35	24	22	15	42	24	5.1	1.6	0	2	0	1.2
Rajani	0.75	0	1.4	2	0	0	1	1	1	4	0	0	2	25	20	17	13	25	16	6		0	1	0	1.7
Sumabenny	0.8	0	1.53	1	0	0	2	1	1	4	2	0	2	35	24	18	15	30	18	5.5		0	1	0	1.6
Mallika	0.45	3	1.45	1	0	0	1	1	1	3	3	0	3	55	20	18	17	60	36	9.3	1	2	1	0	1.4
Jeyanthi	0.79	3	1.75	2	0	0	2	1	1	6	0	0	2	50	22	30	20	50	36	4.2	1.9	2	1	0	1.9
Leena V	0.9	1	1.5	2	0	0	2	1	1	5	2	0	2	35	20	18	15	35	22	3.6	1.6	0	1	0	1.5
Esakkiammal M	0.8	3	1.85	1	0	0	2	1	1	8	0	0	2	30	16	20	15	36	20	7.3	1.5	0	1	0	1.5
Amaravathi	0.94	3	1.45	2	0	0	1	1	1	6	0	0	2	55	12	18	12	65	40	5	1.7	3	1	0	1.4
Selvakumari	0.72	2	1.45	3	1	1	1	1	1	6	0	0	2	35	25	22	19	46	25	9.3	1.3	1	2	0	1.2
Sathyabhama	0.8	1	1.35	2	0	0	1	1	1	7	0	0	0	NA	NA	NA	8	38	22	3	1.5	0	NA	NA	NA
Solly john	0.48	2	1.78	1	0	0	2	1	1	3	0	0	2	35	10	14	9	50	28	6	1.3	1	na	na	na
Joseph mary	0.58	1	1.3	3	1	1	1	1	1	8	0	0	2	35	18	24	21	58	25	3	1.2	1	1	0	1.3
Sheeba	0.61	3	1.5	1	0	0	2	1	1	6	2	0	2	45	16	18	14	52	42	5.4	1.4	3	1	0	1.5
Manju T	NA	0	1.4	2	0	0	1	1	1	6	2	0	2	NA	8	10	8	24	16	NA	NA	0	NA	NA	NA
Shakeela	0.9	1	1.35	0	0	0	1	0	0	8	0	0	1	23	10	10	8	24	12	16	1.2	0	1	0	1.4
Mini kunjumon	0.8	1	1.75	0	0	0	2	1	1	6	2	0	1	27	26	28	20	40	24	3.8	1.7	0	NA	NA	NA
Shamia begum	0.84	1	1.28	2	0	0	1	1	1	9	2	0	1	30	22	22	20	40	24	10	1.2	0	1	0	1.2
Prema S	0.63	2	1.2	2	0	0	1	0	0	6	0	0	2	50	22	19	13	54	26	5.1	1.2	1	2	0	1.2
Uma C	1.2	0	1.6	2	0	0	2	1	1	5.5	0	0	2	30	21	22	17	24	18	4.3	2.7	0	1	0	1.5
Sheema	1.05	1	1.4	3	1	1	1	1	0	10	0	0	2	35	26	28	23	37	24	11	1.5	0	NA	NA	NA
Jyothi laxmi	NA	3	1.76	2	0	0	2	1	1	6	0	0	3	55	18	22	18	33	25	NA	NA	1	NA	NA	NA
Sakunthala MS	NA	3	1.75	2	0	0	2	1	1	5	2	0	2	35	20	20	14	50	26	NA	NA	1	2	0	1
Chandri KC	0.92	2	1.8	2	0	0	2	1	1	3	0	0	1	NA	26	28	20	36	26	11	1.6	1	1	0	1.7
Sheeja tito	0.87	0	1.8	2	0	0	2	1	1	2	0	0	2	25	20	12	11	25	15	3.8	1.4	0	1	0	1.5
Maya	1.47	1	1.75	1	0	0	2	1	1	4.6	0	0	1	22	22	20	19	36	22	4.6	2.3	0	1	0	1.9
Kadeeeja nishac	0.83	3	1.8	1	0	0	2	1	1	3	0	0	1	50	18	22	12	54	38	10	1.2	2	1	0	1.7
Pushpalatha	0.48	3	1.4	2	0	0	1	1	1	4	2	0	2	37	21	18	14	50	25	5	1.1	1	1	0	1.6
Shijina	0.94	2	1.7	2	0	0	2	1	1	5	2	1	2	28	20	15	12	32	18	7.2	1.5	0	NA	NA	NA
Mariamma	0.4	3	1.65	1	0	0	2	1	1	4	0	0	2	34	20	18	16	40	28	13	0.9	1	NA	NA	NA
Sathya G	1.05	2	1.4	3	1	1	1	0	0	5	0	0	2	28	20	30	20	47	27	5.5	1.6	1	1	0	1.3
Sarala P	0.62	3	1.5	1	0	0	2	1	1	10	1	0	2	40	18	16	12	35	25	6.2	1.1	1	1	0	1.3

Maya V	0.75	2	1.63	2	0	0	2	1	1	4	0	0	2	38	23	16	13	36	20	6	1.3	0	1	0	1.5
Mini johnsosn	1.2	1	1.37	2	0	0	1	1	1	4	2	0	2	30	14	12	7	34	18	4	2.4	0	1	0	2
Girija	1.13	0	1.7	1	0	0	2	1	1	4	2	0	1	20	12	10	7	20	12	7	1.7	0	1	0	1.6
Smitha Vinod	0.79	1	1.2	2	0	0	1	0	0	9	0	0	2	30	16	18	13	32	25	4.8	1.3	1	1	0	1.3
Shanmuga khan	0.62	3	1.5	2	0	0	2	1	1	10	2	0	2	45	16	13	12	30	22	14	1	0	NA	NA	NA
Kalaivani N	0.84	1	1.5	2	0	0	2	1	1	6	2	1	2	30	24	12	19	30	18	4	1.5	0	1	0	1.5
Geetha A	0.4	3	1.1	2	0	0	1	1	1	6	0	0	2	50	19	19	16	70	44	9	0.9	3	NA	NA	NA
Nisha ph	na	1	1.4	1	0	0	1	1	1	7	0	0	1	25	NA	NA	15	na	na	na	na	0	1	0	1.4
Mumtaz	0.7	1	1.35	2	0	0	1	1	1	7	0	0	1	25	18	17	16	48	23	6	1.2	0	1	0	1.3
Lakshmi	0.8	1	1.6	2	0	0	2	1	1	2	0	0	1	30	20	14	14	35	23	9	1.1	0	1	0	1.2
Chellam	na	0	1.4	1	0	0	1	1	1	3	2	0	2	25	14	14	12	25	15	na	na	0	NA	NA	NA
Mini G	0.8	0	1.25	2	0	0	1	1	1	6	0	0	1	30	19	14	11	35	18	6	1.3	0	NA	NA	NA
Syed Rabiya	0.75	3	1.35	2	0	0	1	1	1	4	0	0	1	28	18	22	16	30	20	7	1.3	0	1	0	1.3
Thangam P	0.8	3	1.45	2	0	0	1	1	1	5	0	0	2	65	18	16	14	60	39	2	1.4	2	NA	NA	NA
Rajani C	0.95	3	1.4	2	0	0	1	1	1	3	0	0	2	35	10	12	7	35	22	4	1.4	0	NA	NA	NA
Selvalxmi	0.9	0	1.5	2	0	0	2	1	1	11	0	0	2	35	28	26	18	34	18	12	1.1	0	1	0	1.8
Sarithamol	1.2	0	1.7	2	0	0	2	1	1	4	2	1	2	25	18	15	15	36	22	6.3	1.6	0	1	0	1.6
Shibi pappu	1	0	1.4	1	0	0	1	0	0	6	0	0	2	25	10	9	8	25	14	4	1.4	0	NA	NA	NA
Mary pushpa	0.8	0	1.3	1	0	0	1	1	1	6	0	0	1	24	17	14	8	30	18	4	1.3	0	NA	NA	NA
josina joby	na	3	1.35	2	0	0	1	1	1	4	0	0	2	35	28	24	22	na	na	na	na	1	i	0	1.7
Packiyalaxmi	0.57	3	1.4	2	0	0	1	1	1	3	0	0	3	45	12	12	8	40	26	6	1.6	1	1	0	1.5
Sareena Misthaf	1.2	2	1.7	1	0	0	2	1	1	5	0		1	30		19	13	33	17	6.5	2.1	0	2	1	1.8
Jameela	1.1	0	2.15	2	0	0	2	1	1	3	0		1	35	12	9	5	25	12	2	3.6	0	1	0	2
Ramadevi	0.45	2	1.75	1	0	0	2	1	1	5	0		2	40	27	24	18			6	1.1	1	1	0	1.8
Pechiamma	0.45	3	1.7	2	0	0	2	1	1	6	0		1	44	24	22	13	53	34	7.7	1.2	2	1	0	1.7
Leelamma Pius	na	0	1.85	1	0	0	2	1	1	4	0		1	38	14	13	11	24	12	2.7	2	0	1	0	2
Bindhu	0.85	3	1.65	0	0	0	2	1	1	5	0		2	40	24	20	16	50	35	3	1.8	2	1	0	1.7
Vanaja	1.17	1	1.55	0	0	0	2	0	1	4	0		2	40	23	17	14	32	21	6.5	1.3	0	1	0	1.6
Lissy	0.45	3	1.6	3	1	1	2	1	1	6	0		2	68	25	44	26	75	55	21	1	3	1	1	1.6

INTERIM FU						FINAL FOLLOW UP													
name	MS Mean	MR	TR	RVSP	PAH grade(0-none, 1-mild, 2- moderate, 3- seve	Functional class	Rhythm(0-sr, 1-af)	MVA (Avg)	MS Mean	MR	AR	AS	TR	RVSP	PAH grade(0-none, 1-mild, 2- moderate, 3- seve	RESTENOSIS	Re interventions (0- NO, 1- YES, 2- PLANNED	H/o. SUB BMV (0-no, 1- yes)	H/o. SUB MVR (0-no, 1- yes)	Time to Last Follow up (YRS)
Hancya.A.A	6	2	2	46	2	1	0	1.5	11	2	3	3	2	35	1	0	0	0	0	6
Prabha.V	4	1	1	26	0	2	0	1.4	6	3	0	0	3	48	2	0	0	0	0	1
Murugesari.B	4	1	1	30	1	2	0	1.2	5	2	3	2	2	39	1	1	0	0	0	1
Thiru selvi	4	2	2	39	1	2	0	0.9	10	1	2	2	2	56	2	1	0	0	0	1
Deepika	11	2	2	48	2	1	0	1.8	2	2	3	2	2	40	1	0	0	0	0	1
Suma.A.	4	1	1	29	0	1	0	1.6	7	2	0	0	2	36	1	0	0	0	0	6
Mnju .S	6	2	2	40	1	1	0	1.4	6	3	0	0	2	50	2	0	0	0	0	4
Sakkala K	NA	NA	NA	NA	na	2	1	1.3	16	2	3	3	2	55	2	1	1	0	1	5
Selvi	5	2	2	55	2	1	0	1.8	4	2	1	0	2	46	2	0	0	0	0	4
Anie	12	3	3	58	2	2	1	2.4	18	3	0	0	2	55	2	0	0	0	0	6
Seenath	4	1	2	40	1	1	0	1.9	3	0	2	0	2	35	1	0	0	0	0	5
Marammal	4	1	1	40	1	1	0	1.3	9	1	0	0	2	40	1	0	0	0	0	4
Elsy Binny	4	1	2	35	1	1	0	2.1	4	1	1	0	1	26	0	0	0	0	0	5
Haleema K	3	2	1	28	0	1	0	1.7	7	1	0	0	1	30	1	0	0	0	0	6
Mini Haridas	7	1	2	38	1	1	0	2.3	3	1	0	0	1	24	0	0	0	0	0	6
Sudha A	4	2	2	32	1	1	0	1.3	8	1	0	0	1	25	0	0	0	0	0	6
Rajalakshmi	4	2	1	26	0	1	0	2	3	2	1	0	1	30	1	0	0	0	0	5
Radhika	7	2	2	34	1	1	0	1.5	6	2	2	0	2	41	1	0	0	0	0	2
Rasheeda	4	0	NA	NA	na	1	0	1.8	6	2	2	0	2	34	1	0	0	0	0	2
Chithayee M	6	2	2	37	1	1	0	1.5	8	1	2	0	2	40	1	0	0	0	0	6

Shailaja	8	2	2	43	1	1	0	1.7	3	2	3	0	3	45	1	0	0	0	0	3
Jaisy K	10	2	2	41	1	1	0	1.3	11	2	2	1	2	34	1	0	0	0	0	7
Alma	6	1	2	35	1	1	0	1.5	9	1	1	0	1	38	1	0	0	0	0	7
Sudha C	4	2	2	45	1	1	0	1.9	5	2	3	1	2	46	2	0	0	0	0	6
Radha B	6	2	2	26	0	1	0	1.3	6	1	0	2	1	25	0	0	0	0	0	6
Amutha	4	1	2	44	1	1	0	1.8	4	2	2	0	2	44	1	0	0	0	0	5
Usha G	3	1	0	NA	na	1	0	1.8	5	1	3	0	1	32	1	0	0	0	0	4
Bijiamma G	12	1	1	48	2	2	0	0.8	10	0	0	0	2	54	2	1	1	0	1	5
Devu P.K.	3	1	0	NA	na	1	0	1.4	9	2	2	0	0	NA	NA	0	0	0	0	10
Anitha	3	1	1	NA	na	1	0	1.6	6	2	0	0	1	55	2	0	0	0	0	10
Rajesswari	7	0	1	59	2	1	0	1.1	24	0	0	0	2	50	2	0	0	0	0	8
Honey	5	1	2	46	2	1	0	1.4	8	2	2	0	0	NA	NA	1	1	1	0	10
Ummukulusu	6	2	2	50	2	1	0	1.4	4	1	0	0	1	38	1	0	0	0	0	5
Latha	4	2	2	40	1	1	0	1.8	7	2	0	0	2	50	2	0	0	0	0	5
Shahinsha Mol	8	1	2	58	2	2	0	1.3	15	1	2	0	2	60	3	0	0	0	0	4
Somy sunny	4	0	1	28	0	1	0	1.8	4	1	0	0	2	32	1	0	0	0	0	4
Jebarlin	5	1	1	37	1	1	0	1.3	6	0	1	0	2	35	1	0	0	0	0	4
Ponnammai S	3	0	2	34	1	1	0	1.8	4	0	0	0	1	28	0	0	0	0	0	4
Safeera	10	2	2	60	3	1	0	1.2	11	2	1	0	2	56	2	1	0	0	0	2
Hema	16	1	3	56	2	1	0	1.4	9	0	0	0	2	40	1	0	0	0	0	5
Kunjamma Varghe	4	1	2	58	2	2	0	1.5	6	1	1	0	2	50	2	0	0	0	0	5
Subaida Moidu	3	2	2	55	2	1	0	1.6	6	3	3	0	2	55	2	0	0	0	0	5
Shiny	4	1	2	34	1	2	0	1	23	2	3	0	2	40	1	1	0	0	0	9
Sjithra	5	2	2	50	2	1	0	1.8	7	1	0	0	2	36	1	0	0	0	0	6
Lissy KM Zacharia	6	0	2	40	1	1	0	1.4	8	0	2	0	2	36	1	0	0	0	0	4
Aysha Bai	6	0	0	NA	na	1	0	1.4	5	2	2	0	2	34	1	0	0	0	0	8
Simi L	6	2	2	34	1	1	0	2	6	2	2	0	2	40	1	0	0	0	0	7
Christy	6	1	2	56	2	1	0	1.8	6	2	2	0	2	50	2	0	0	0	0	7
Sindhu KP	4	1	2	31	1	1	0	1.8	3	2	0	0	2	28	0	0	0	0	0	6
Sheela	6	3	2	74	3	1	0	1.3	4	2	0	0	2	50	2	0	0	0	0	6
Geetha	6	2	2	37	1	1	0	1.7	3	2	0	0	2	30	1	0	0	0	0	8
Tresa L	6	1	2	40	1	1	0	1.3	5	1	3	0	2	28	0	1	0	0	0	7
Usha S	18	3	2	87	3	2	1	1.1	10	3	0	0	3	58	2	0	0	0	0	4
Sheeja A	3	2	1	32	1	1	0	1.7	4	2	0	0	1	28	0	0	0	0	0	3
Soudha K	3	1	1	25	0	1	0	1.5	5	1	0	0	1	25	0	0	0	0	0	3
Aneesha	9	3	3	55	2	2	0	1.1	10	3	0	0	3	55	2	0	0	0	0	3
Selva sundari	5	0	1	35	1	2	0	1.3	6	0	0	0	1	35	1	0	0	0	0	2.5
Anitha k	6	2	3	45	1	2	0	1.3	8	2	0	0	3	48	2	0	0	0	0	5

Esakkiammal s	7	3	2	30	1	2	0	1.4	8	3	3	0	2	30	1	0	0	0	0	3
Naseema beevi	8	1	2	40	1	2	0	0.9	16	1	0	0	2	50	2	1	1	1	0	5
USHA K	4	2	2	45	1	2	0	1.4	5	2	3	0	2	44	1	1	1	0	1	4
Balamani	5	2	1	25	0	2	0	1.1	4	2	2	0	1	25	0	0	0	0	0	4
Rajani	4	2	2	25	0	2	0	1.3	6	2	0	0	2	45	1	0	0	0	0	5
Sumabenny	4	1	2	25	0	1	0	1.6	4	1	2	0	2	25	0	0	0	0	0	4
Mallika	4	1	2	50	2	2	0	1.4	5	1	3	0	3	50	2	0	0	0	0	2
Jeyanthi	4	2	2	25	0	1	0	1.8	6	2	0	0	2	25	0	0	0	0	0	5
Leena V	6	2	2	35	1	1	0	1.5	7	2	2	0	2	44	1	0	0	0	0	4
Esakkiammal M	9	1	2	25	0	2	0	1.1	14	1	0	0	2	30	1	1	0	0	0	5
Amaravathi	8	2	2	55	2	2	0	1.4	9	2	0	0	2	50	2	0	0	0	0	4
Selvakumari	7	3	2	25	0	2	0	1.1	8	3	0	0	2	25	0	0	0	0	0	4
Sathyabhama	NA	NA	NA	NA	na	2	0	1.2	9	2	0	0	0	NA	NA	0	0	0	0	2
Solly john	na	na	na	na	na	1	0	1.5	7	2	0	0	2	40	1	0	0	0	0	1.3
Joseph mary	9	3	0	40	1	2	0	1.3	9	3	0	0	2	35	1	0	0	0	0	2
Sheeba	8	1	2	45	1	2	0	1.4	14	1	3	1	2	48	2	0	0	0	0	3
Manju T	NA	NA	NA	NA	na	1	0	1.3	8	2	2	0	0	NA	NA	0	0	0	0	1
Shakeela	8	0	0	NA	na	1	0	1.5	4	0	0	0	0	NA	NA	0	0	0	0	2
Mini kunjumon	NA	NA	NA	NA	na	1	0	1.6	7	0	2	0	0	25	0	0	0	0	0	2
Shamia begum	10	2	2	45	1	1	0	1.2	10	2	2	0	2	50	2	0	0	0	0	3
Prema S	9	2	2	55	2	2	0	0.8	16	2	0	0	2	60	3	1	1	1	0	4
Uma C	6	2	2	30	1	1	0	1.4	8	2	0	0	2	35	1	0	0	0	0	3
Sheema	NA	NA	NA	NA	na	1	0	1.4	15	3	0	0	2	30	1	0	0	0	0	1
Jyothi laxmi	NA	NA	NA	NA	na	1	0	1.6	7	2	0	0	3	45	1	0	0	0	0	1
Sakunthala MS	22	2	2	70	3	1	0	1.8	8	2	2	0	2	40	1	1	1	1	0	2.5
Chandri KC	4	2	1	NA	na	2	1	1.6	6	2	2	0	2	28	0	0	0	0	0	2
Sheeja tito	7	2	1	15	0	2	0	1.4	8	2	0	0	1	20	0	0	0	0	0	2
Maya	3	2	1	20	0	1	0	1.7	5	2	0	0	1	24	0	0	0	0	0	3
Kadeeeja nishad	4	1	1	37	1	1	0	1.7	7	1	0	0	1	30	1	0	0	0	0	4
Pushpalatha	5	2	2	25	0	1	0	1.5	6	2	2	0	2	35	1	0	0	0	0	3
Shijina	NA	NA	NA	NA	na	2	0	1.5	7	2	2	1	2	25	0	0	0	0	0	3
Mariamma	NA	NA	NA	NA	na	2	0	1.4	7	2	0	0	2	35	1	0	0	0	0	4
Sathya G	6	3	2	25	0	1	0	1.4	8	3	0	0	2	25	0	0	0	0	0	3
Sarala P	4	1	2	30	1	1	0	1.2	5	1	3	0	2	30	1	0	0	0	0	4

Maya V	7	2	2	36	1	1	0	1.3	8	2	2	1	2	25	0	1	0	0	0	3.75
Mini johnsosn	4	2	2	25	0	1	0	1.9	5	2	2	0	2	25	0	0	0	0	0	3
Girija	4	1	1	25	0	1	0	1.4	4	1	2	0	1	25	0	0	0	0	0	3.25
Smitha Vinod	7	2	2	30	1	1	0	1.1	9.8	2	0	0	2	35	1	0	0	0	0	3.5
Shanmuga khani	NA	NA	NA	NA	na	1	0	1.4	9	2	2	0	2	25	0	0	0	0	0	0.75
Kalaivani N	9	2	2	25	0	2	0	1.3	11	2	3	1	2	30	1	0	0	0	0	3
Geetha A	NA	NA	NA	NA	na	2	0	1.1	9	2	0	0	2	50	2	0	0	0	0	1.25
Nisha ph	7	2	1	25	0	1	0	1.3	8	2	0	0	1	30	1	0	0	0	0	2
Mumtaz	7	2	1	25	0	1	0	1.2	9	2	0	0	2	30	1	0	0	0	0	1.5
Lakshmi	15	2	2	45	1	2	0	1	13	2	0	0	2	40	1	1	1	1	0	2.5
Chellam	NA	NA	NA	NA	na	1	0	1.3	5	2	2	0	2	25	0	0	0	0	0	0.5
Mini G	NA	NA	NA	NA	na	1	0	1.2	7	2	0	0	1	25	0	0	0	0	0	0.5
Syed Rabiya	7	2	1	25	0	1	0	1.4	7	2	0	0	1	20	0	0	0	0	0	0.75
Thangam P	NA	NA	NA	NA	na	1	0	1.2	5	2	2	0	2	45	1	0	0	0	0	0.75
Rajani C	NA	NA	NA	NA	na	1	0	2	5	2	0	0	2	45	1	0	0	0	0	0.75
Selvalxmi	9	2	2	40	1	1	0	1.4	10	2	0	0	2	35	1	0	0	0	0	1.5
Sarithamol	10	2	2	26	0	2	0	1.3	12	2	2	1	1	18	0	1	0	0	0	1.5
Shibi pappu	NA	NA	NA	NA	na	1	0	1.5	9	2	0	0	2	25	0	0	0	0	0	0.75
Mary pushpa	NA	NA	NA	NA	na	1	0	1.2	8	2	0	0	2	24	0	0	0	0	0	1
josina joby	5	2	2	30	1	1	0	1.5	5	2	0	0	2	35	1	0	0	0	0	1.5
Packiyalaxmi	7	2	3	25	0	1	0	1.4	8	2	0	0	3	30	1	0	0	0	0	1.5
Sareena Misthafa	4	1	1	30	1	2	1	1.3	11	1	1	0	2	37	1	0	0	0	0	11
Jameela	3	2	1	35	1	1	0	2.1	3	1	0	0	1	30	1	0	0	0	0	6
Ramadevi	45	1	2	36	1	1	0	1.5	4	1	0	0	1	30	1	0	0	0	0	3
Pechiamma	4	2	1	46	2	1	0	1.4	6	2	0	0	2	46	2	0	0	0	0	8
Leelamma Pius	3	1	0		na	1	0	1.3	8	1	0	0	2	25	0	1	0	0	0	1
Bindhu	4.5	0	2	40	1	1	0	1.2	12	1	0	0	3	50	2	1	1	1	0	14
Vanaja	4	0	2	40	1	1	0	1.4	6	2	1	0	1	48	2	1	1	1	0	13
Lissy	7	3	3	69	3	na	na	na	na	na	na	na	na	na	na	NA	1	0	1	1

DEMOGRAPHICS																	Pre procedure												POST PROCI			
Name	HospitalNO	Age	H/o RF(0- Absent, 1-Present)	Diagnosis of RHD(1-PRESENT PREGNANCY, 2- DIAGNOSED PREVIOUSLY)	Date of ptmc	interv type(0-none, 1- cmv, 2-ptmc, 3- multiple	pregnancies (Number)	Children (Number)	ABORTIONS (0- NO, 1- YES)	MTP(0- NO, 1- YES)	Functional class (0- NO, 1- YES)	Rhythym (0- SR, 1-AF)	H/O CCF (0- NO, 1- YES)	H/O Embolism (0- none, 1-TIA, 2-CVA, 3-peripheral)	Gestational age at PTMC (weeks)	Fluoro time(mins)	PRELAa	PreLAv	PreLAm	PreMSgradient	PreMVA	PreRVSP	PreMR	PreAS	PreAR	PreTR	PAH GRADE (0- NO, 1-MILD, 2-MOD, 3-SEVEERE	PostLAa	PostLAv	PostLAm	PostMSgradient
sindhu s	249389	25	0	1	8/2/2006	0	0	0	0	0	3	0	0	0	32	4.5	20	20	16	22	0.9	50	2	0	0	2	2	6	6	5	5
Geeta m	189740	23	0	1	21/01/2001	0	1	1	0	0	3	0	0	0	24	5.5	15	24	20	14	1.05	55	1	0	0	2	2	7	12	8	5
Jacknu bab	304714	27	0	1	9/12/2009	0	1	1	0	0	3	0	0	0	26	6	34	40	34	27	0.8	120	2	0	2	2	3	24	36	20	18
sudharani	250344	30	0	1	10/3/2006	0	1	1	0	0	2	0	0	0	24	na	50	50	38	28	0.8	110	2	0	0	2	3	28	28	16	5
Rajani R	219837	21	0	1	11/10/2003	0	0	0	0	0	3	0	0	0	30	na	34	38	32	29	0.8	110	1	0	1	3	3	16	18	14	14
Ambili NS	227036	32	0	2	24/06/2004	0	2	0	2	0	3	0	0	0	24	13	36	36	25	18	0.8	90	1	0	0	3	3	18	18	13	10
Geoselva	258170	27	0	1	16/12/2006	0	1	1	0	0	2	0	0	0	28	na	36	32	35	29	1	90	2	0	0	3	3	22	20	18	4
Jalaja s	197553	21	0	2	9/12/2001	0	0	0	0	0	2	0	0	0	24	2.1	46	30	30	20	0.8	25	2	0	0	1	0	24	26	20	5
christy t	235271	25	0	1	17/03/2005	0	0	0	0	0	2	0	0	0	30	na	30	26	24	26	0.7	65	2	0	0	2	3	16	14	13	10
sasikala	220595	29	0	2	18/10/2003	0	1	1	0	0	2	0	0	0	34	12	30	40	29	29	0.5	115	2	0	0	3	3	30	52	22	12
Sudhimol	276266	19	0	1	30/01/2008	0	0	0	0	0	3	0	0	0	28	1.45	28	24	22	20	0.9	58	2	0	0	2	2	18	15	15	9
Malathi	300500	23	0	1	14/09/2009	0	1	1	0	0	2	0	0	0	34	na	40	56	39	22	0.8	80	2	0	0	3	3	20	20	14	8
Rajani MK	8707175	34	0	2	20/03/2002	1	0	0	0	0	3	0	0	0	28	4	26	40	29	20	0.85	65	2	0	0	2	3	18	29	20	6
kumari PK	237685	27	1	2	1/1/2008	0	0	0	0	0	2	0	0	0	24	4.5	48	40	40	20	0.9	80	2	0	0	2	3	24	20	18	8
Jain G	225459	25	0	2	24/06/2004	0	1	1	0	0	3	0	0	0	28	na	32	33	26	24	0.75	100	2	0	0	3	3	16	18	10	3
Suja r	9303771	21	0	2	10/7/2006	1	0	0	0	0	3	0	0	0	28	6	55	45	41	28	1.1	75	2	0	0	3	3	32	30	22	13
Sainaba S	9409103	32	1	2	17/12/2007	2	0	0	0	0	2	0	0	1	26	4.4	38	58	38	22	0.9	65	1	1	2	3	3	24	25	18	9
Petchiamm	301886	29	0	2	5/10/2009	0	0	0	0	0	2	0	0	0	32	na	36	44	30	18	0.95	80	2	0	0	3	3	24	22	24	10
Mary TP	8900376	39	1	2	22/07/2006	0	0	0	0	0	2	0	0	0	28	4	31	40	32	20	0.9	35	2	0	0	2	1	30	26	26	9
Nandini KP	238059	30	0	2	6/6/2005	0	1	1	0	0	2	0	0	0	28	na	32	34	18	14	1.05	45	2	0	0	2	2	13	13	10	4
Sheela L	9901741	24	0	1	23/07/1999	0	0	0	0	0	2	0	0	0	32	5	30	30	22	18	0.7	45	2	1	3	2	2	12	12	6	8

Ramla T	198856	23	0	2	8/1/2002	0	2	2	0	0	3	0	0	0	12	4.5	20	20	12	22	1	46	2	0	0	2	2	13	10	6	6
Bindu S	242908	28	1	2	11/8/2005	0	1	0	1	0	2	0	0	0	24	3	30	38	20	16	0.95	40	2	0	3	2	1	20	24	12	9
Ammini Ra	187591	22	0	2	10/2/2002	0	1	1	0	0	2	0	0	0	26	4	32	28	31	16	1	35	1	0	2	2	1	25	26	20	6
Shemi niss	260403	24	0	1	27/12/2006	0	1	1	0	0	2	0	0	0	32	4	26	24	16	18	0.8	48	2	0	0	2	2	14	12	8	6
Geetha	200203	25	0	1	6/5/2002	0	0	0	0	0	3	0	0	0	26	3	28	30	20	34	0.7	28	2	0	0	1	0	16	20	14	8
Anitha K	193880	24	0	2	7/9/2001	0	1	1	0	0	3	0	0	0	26	4.5	42	36	32	14	0.6	50	1	0	0	2	2	17	13	12	5
Jannath	38988	28	0	2	17/09/2000	1	2	1	1	0	2	0	0	0	28	4	35	30	26	19	0.6	50	1	0	0	2	2	20	18	16	10
Ponnamm	272087	27	0	1	16/11/2007	0	0	0	0	0	3	0	0	0	28	1	40	70	45	21	1	75	3	0	0	3	3	26	38	24	7
Remya Ma	280120	20	0	1	28/04/2008	0	0	0	0	0	3	0	0	0	30	5	32	36	24	26	1	65	1	0	0	2	3	20	17	13	8
Khadeeja v	247652	35	1	2	1/12/2009	2	1	1	0	0	2	0	0	0	28	3	33	44	21	19	0.7	45	2	0	0	2	2	18	18	16	10
Biindu Dee	248095	25	0	1	10/1/2006	0	0	0	0	0	2	0	0	0	32	2.3	20	18	18	22	0.85	86	2	0	0	3	3	12	8	8	10
Sreekala.T	217328	22	0	2	25/08/2003	0	0	0	0	0	3	0	0	0	24	na	27	38	26	21	0.9	45	2	0	0	2	2	20	24	16	11
Deena DN	201792	20	0	2	20/03/2002	2	0	0	0	0	3	0	0	0	26	1.5	20	15	14	33	0.9	35	2	0	0	1	1	18	11	10	14
Anjana	189289	27	1	2	29/01/2001	0	0	0	0	0	2	0	0	0	26	3.5	28	26	21	16	0.9	55	1	0	0	2	2	20	26	17	8
Rajani TB	9206161	32	1	2	18/05/2005	0	0	0	0	0	3	0	0	0	29	3	40	38	30	20	0.95	65	2	0	2	2	3	26	10	8	10
Thahira PJ	247393	27	0	1	27/11/2005	0	1	1	0	0	2	0	0	0	26	4	24	16	15	11	0.85	38	1	0	0	1	1	11	8	8	7
Sandhya c	232200	22	0	1	8/10/2004	0	1	1	0	0	2	0	0	0	30	3	30	32	32	13	0.9	44	2	0	0	1	1	12	10	9	6
Sujath Bee	237082	23	0	1	20/02/2005	0	2	2	0	0	2	0	0	0	28	4.5	24	26	20	20	1.05	42	2	0	3	1	1	16	10	12	7
Celin S	9707329	32	0	2	14/07/2007	2	1	1	0	0	3	0	0	0	34	2.5	22	21	20	16	0.95	25	2	0	0	1	0	16	11	10	7
Vijaya n	9000179	42	0	2	19/11/2001	0	2	2	0	0	3	0	0	0	29	3.5	40	50	35	24	0.55	110	1	0	0	3	3	10	13	7	6
Jaya S	245984	21	0	1	25/11/2001	0	0	0	0	0	3	0	0	0	24	4	28	32	24	19	1	70	3	0	3	3	3	20	17	14	2
Ambili N	297823	27	0	2	19/10/2009	0	1	1	0	0	2	0	0	0	30	5	32	40	29	19	0.85	70	2	0	0	2	3	20	28	12	7
Murugamm	265803	30	0	2	11/9/2007	0	1	1	0	0	2	0	0	0	30	2	38	40	40	24	0.7	65	2	0	0	2	3	14	8	6	5
Geetha M	298951	24	0	1	22/07/2009	0	2	2	0	0	2	0	0	0	34	na	20	18	16	20	0.9	75	2	0	0	2	3	13	12	10	5
Seena NC	284488	28	0	1	8/8/2008	0	0	0	0	0	3	0	0	0	28	4.5	35	24	28	18	0.8	38	2	0	0	2	1	16	8	10	6
Rajani Kum	224518	31	0	1	19/04/2004	0	1	1	0	0	3	0	0	0	28	3	26	30	24	26	0.8	40	2	0	0	2	1	14	16	12	10
Palthai K	278736	25	0	1	31/3/2008	0	1	1	0	0	3	0	0	0	28	na	na	na	28	23	0.75	70	1	0	0	3	3	na	na	16	10
Metancy P	303334	26	0	1	30/10/09	0	1	1	0	0	2	0	0	0	36	5	26	24	21	21	0.77	70	1	0	2	3	3	16	16	12	9
Shemeera	233940	18	0	2	25.11.04	0	0	0	0	0	2	0	0	0	20	3.5	24	25	21	21	0.65	100	2	1	2	3	3	20	22	17	5
Jameela	9509759	27	0	1	28.12.95	1	0	0	0	0	3	0	0	0	20	4	na	na	29	37	0.85	66	1	0	0	2	3	na	na	22	10
Noorjahan	199384	20	0	1	7.01.02	0	0	0	0	0	2	0	0	0	32	5	20	30	25	16	1	46	2	0	3	2	2	12	16	11	14
Siva Jothi	198910	31	1	2	31.12.01	1	1	1	0	0	3	1	0	0	24	4.5	30	42	30	18	0.8	108	0	0	2	2	3	18	22	17	10
Reena K	9505097	26	0	1	28.8.95	0	0	0	0	0	3	0	0	0	26	4.5	40	38	32	15	0.95	70	1	0	0	2	3	24	18	16	11
Kaliammal	9803732	23	1	1	6.8.98	0	0	0	0	0	2	0	0	0	36	3.5	30	25	20	10	0.7	90	0	0	0	4	3	20	22	18	4
Bidhu madl	9709003	24	0	1	1.1.98	0	0	0	0	0	2	0	0	0	24	2	50	62	42	25	0.7	70	0	0	1	2	3	23	20	12	4
Girija	9601054	21	1	1	16.2.96	0	0	0	0	0	2	0	0	0	34	5	22	19	12	10	0.85	50	2	0	0	2	2	16	17	10	6
Saleena Be	9600195	20	0	1	11.1.96	0	0	0	0	0	3	0	0	0	26	5	30	37	28	28	0.64	NA	2	0	0	0	NA	19	21	15	5
Rethnamm	9509841	35	0	2	18.12.95	0	2	2	0	0	3	0	0	0	28	NA	37	36	30	25	0.85	94	0	0	0	3	3	27	24	15	14
Jayaraniri	9802153	21	0	1	15.10.98	0	0	0	0	0	2	0	0	0	30	3	34	34	25	13	0.85	50	1	2	2	1	2	22	19	14	6
Sarah P	9409918	20	0	1	16.7.02	0	0	0	0	0	2	0	0	0	24	NA	25	31	22	16	1	54	1	0	2	2	2	20	21	18	6
Sandhya P	260935	25	0	1	16.03.07	0	0	0	0	0	3	0	0	0	30	4.8	26	17	15	20	0.9	50	2	0	2	2	2	18	15	12	8
Sarojini k	233119	23	0	2	21/10/04	0	0	0	0	0	4	1	0	0	24	20	24	24	19	30	0.8	125	2	0	0	0	3	12	20	9	12

EDURE					POST PROCEDURE						INTERIM FU								Final follow up														
PostMVA	PostMR	PostAR	PostTR	PostRVSP	MVA Average (0- < 1.5; 1-> = 1.5)	MVA CHANGE > 50% (0- NO, 1- YES)	MR> 3 (0- NO, 1- YES)	Good result(0- NO, 1- YES)	PAH GRADE (0- NO, 1-MILD, 2-MOD, 3-SEVERE	complications (0- none, 1- thromboembolism, 2-mortality	FU Functional class	Rhythym(0-SR, 1-AF)	FUMSgradient	FUMVA	FUMR	FUTR	FURVSP	PAH GRADE (0- NO, 1-MILD, 2-MOD, 3-SEVEERE	MaternalFC	FU Rhythym (0- SR, 1-AF)	LastMSgradient	LastMVA	LastMR	LastAS	LastAR	LastTR	LastRVSP	PAH GRADE (0- NO, 1-MILD, 2-MOD, 3-SEVEERE	Subsequent Pregnancies (number)	subsequent delivery (Number)	RESTENOSIS	Subsequent number of interventions	FOLLOW UP (YEARS)	Subsequentinterventions (1- cmv, 2-ptmc, 3- MVR,
1.5	2	0	2	25	1	1	0	1	0	0	1	0	8	1.3	2	2	25	0	2	0	9	1.2	2.5	0	0	2	20	0	0	0	1	0	4.42	0
1.5	2	0	2	30	1	0	0	1	1	0	1	0	6	1.7	2	0	23	0	1	0	6	1.6	2	0	0	2	28	0	0	0	0	0	9.50	0
1.45	3	2	2	55	0	1	1	1	2	0	1	0	9	1.4	3	2	60	3	2	0	8	1.45	3	0	2	2	53	2	0	0	0	0	0.58	0
1.3	2	0	2	55	0	1	0	1	2	0	1	0	8	1.2	2	2	45	2	1	0	9	1.1	2.5	0	0	2	35	1	0	0	0	0	4.33	0
1.55	2	1	2	65	1	1	0	1	3	0	1	0	11	1.4	2	2	40	1	2	0	9	1.1	2	0	2	2	40	1	1	1	0	0	9.58	0
1.5	1	0	2	85	1	1	0	1	3	0	1	0	9	1.5	1	2	68	3	1	1	9	1.2	1	0	0	2	35	1	0	0	0	0	3.80	0
2	2	0	2	40	1	1	0	1	1	0	1	0	5	1.8	2	2	25	0	1	0	6	1.7	2	0	0	2	20	0	0	0	0	0	3.58	0
1.46	2	0	1	20	0	1	0	1	0	0	1	0	6	1.4	2	2	20	0	1	0	9	1.3	2	0	0	2	20	0	1	1	0	0	9.58	0
1.6	2	0	2	40	1	1	0	1	1	0	1	0	8	1.6	2	2	40	1	2	0	9	1.4	2.5	0	2	2	35	1	1	1	0	0	5.30	0
1.45	3	0	3	68	0	1	1	1	3	0	1	0	8	1.5	3	2	35	1	1	0	12	1.15	3.5	0	0	2	33	1	0	0	0	0	6.75	0
1.6	2	0	2	45	1	1	0	1	2	0	1	0	8	1.5	3	2	38	1	2	0	10	1.2	3	0	0	2	35	1	1	1	0	0	2.42	0
1.55	2	1	2	40	1	1	0	1	1	0	1	0	5	1.6	2	2	35	1	1	0	5	1.5	2.5	0	2	2	35	1	0	0	0	0	8.30	0
1.1	2	0	2	55	0	0	0	0	2	0	1	0	7	1	2	2	35	1	2	0	12	1.1	2	0	0	2	40	1	0	0	1	1	1.00	2
1.7	2	0	2	40	1	1	0	1	1	0	1	0	8	1.6	2	2	35	1	1	0	6	1.55	2	0	0	2	35	1	0	0	0	0	2.50	0
2	2	0	2	25	1	1	0	1	0	0	1	0	4	1.8	2	2	25	0	1	0	5	1.75	2	0	0	1	25	0	0	0	0	0	6.10	0
1.4	2	0	2	55	0	0	0	0	2	0	1	0	10	1.4	2	2	40	1	2	0	12	1.2	2	0	0	2	35	1	0	0	0	0	4.00	0
1.6	1	2	2	35	1	1	0	1	1	0	1	0	5	1.4	1	2	25	0	1	0	6	1.3	1	1	3	2	25	0	0	0	0	0	2.58	0
1.3	2	0	2	75	0	0	0	0	3	0	1	0	6	1.3	2	2	26	0	1	0	8	1.2	2	0	0	2	28	0	0	0	0	0	0.75	0
1.7	2	0	2	25	1	1	0	1	0	0	1	0	7	1.8	2	2	22	0	1	0	7.5	1.65	2	0	0	2	20	0	0	0	0	0	4.00	0
1.55	2	0	2	25	1	1	0	1	0	0	1	0	4	1.6	2	2	25	0	1	0	5	1.5	2	0	0	2	22	0	0	0	0	0	5.08	0
1.45	2	3	2	35	0	1	0	1	1	0	1	0	8	1.4	2	2	25	0	2	0	12	1.15	2	2	3	2	25	0	1	1	1	1	6.00	2

1.6	2	0	2	32	1	1	0	1	1	0	1	0	7	1.8	2	2	35	1	2	0	10	1.7	2	0	0	2	35	1	0	0	0	0	8.50	0
1.75	2	3	2	25	1	1	0	1	0	0	1	0	5	1.7	2	2	22	0	2	0	5.5	1.6	2	1	3	2	20	0	0	0	0	0	4.92	0
1.7	1	2	2	25	1	1	0	1	0	0	1	0	5	1.7	1	2	24	0	1	0	4	1.65	2	0	2	2	26	0	0	0	0	0	8.50	0
1.6	2	0	2	28	1	1	0	1	0	0	1	0	7	1.7	2	2	25	0	2	0	8	1.5	2	0	0	2	34	1	0	0	0	0	3.50	0
2.1	2	0	1	25	1	1	0	1	0	0	1	0	8.5	1.6	2	1	22	0	1	0	8	1.54	2	0	0	1	25	0	1	1	0	0	9.20	0
1.56	2	0	2	42	1	1	0	1	1	0	1	0	3	1.7	2	2	35	1	1	0	6	1.6	2	0	0	2	28	0	0	0	0	0	8.83	0
1.35	1	0	1	48	0	1	0	1	2	0	1	0	9	1.4	1	1	35	1	2	0	7.5	1.3	1	0	0	2	35	1	0	0	1	1	7.00	2
1.7	3	0	2	45	1	1	1	1	2	0	1	0	7	1.7	3	2	40	1	1	0	9	1.5	3	0	0	2	38	1	0	0	0	0	3.67	0
1.5	1	0	2	45	1	1	0	1	2	0	1	0	6	1.5	1	2	35	1	1	0	7	1.5	1	0	0	2	28	0	0	0	0	0	2.17	0
1.65	2	0	2	45	1	1	0	1	2	0	1	0	7.5	1.6	2	2	35	1	1	0	9	1.25	2	0	0	2	35	1	0	0	0	0	0.60	0
1.8	3	0	2	55	1	1	1	1	2	0	1	0	7	1.8	3	2	38	1	1	0	5	1.9	3	0	0	2	28	0	0	0	0	0	4.50	0
1.9	2	0	2	35	1	1	0	1	1	0	1	0	6	2	2	2	28	0	1	0	9	1.74	2.5	0	0	2	28	0	1	1	0	0	6.83	0
1.6	2	0	1	32	1	1	0	1	1	0	1	0	12	1.5	2	1	32	1	2	0	10	1.45	2	0	0	1	25	0	1	1	0	0	8.25	0
1.45	2	0	2	40	0	1	0	1	1	0	1	0	5	1.6	2	2	25	0	2	0	11	1.1	2	0	0	2	28	0	1	1	1	1	9.00	2
1.3	3	2	2	58	0	0	1	0	2	0	1	0	8	1.4	2	2	45	2	2	0	5	1.36	2	0	2	2	38	1	0	0	0	0	5.20	0
1.6	2	0	1	35	1	1	0	1	1	0	1	0	8	1.6	2	1	25	0	1	0	8	1.5	2	0	0	1	24	0	0	0	0	0	4.70	0
1.75	2	0	1	45	1	1	0	1	2	0	1	0	4	1.8	2	1	35	1	1	0	5	1.2	2	0	0	1	30	1	0	0	0	0	5.75	0
1.65	2	3	1	35	1	1	0	1	1	0	1	0	5	1.6	2	1	24	0	1	0	10	1.15	2	0	3	1	25	0	0	0	0	0	5.00	0
1.25	2	0	1	24	0	0	0	0	0	0	1	0	8	1.3	2	1	24	0	2	0	11	1.15	2	0	0	1	22	0	0	0	0	0	3.00	0
1.7	1	0	2	56	1	1	0	1	2	0	1	0	4	1.7	1	2	35	1	1	0	8	1.65	1	0	0	2	35	1	0	0	0	0	8.50	0
1.45	3	3	2	55	0	0	1	0	2	0	1	0	6	1.5	3	2	38	1	1	0	5	1.56	3	0	3	2	35	1	2	2	0	0	8.67	0
1.45	2	0	2	35	0	1	0	1	1	0	1	0	5	1.5	2	2	25	0	1	0	6	1.4	2	0	0	2	25	0	0	0	0	0	0.75	0
1.5	2	0	2	55	1	1	0	1	2	0	1	0	4	1.6	2	2	35	1	1	0	6	1.7	1	0	0	2	25	0	0	0	0	0	2.80	0
1.4	2	0	2	30	0	1	0	1	1	1	1	0	5	1.5	2	2	25	0	1	0	6	1.3	2	0	0	2	25	0	0	0	0	0	1.00	0
1.35	2	0	2	35	0	1	0	1	1	0	1	0	6	1.4	2	2	25	0	1	0	7	1.3	2	0	0	2	25	0	0	0	0	0	2.00	0
1.58	3	0	2	25	1	1	1	1	0	0	1	0	7	1.4	3	2	25	0	2	0	5	1.4	3	1	3	2	25	0	0	0	0	0	6.25	0
1.22	1	0	3	55	0	1	0	1	3	0	NA	NA	na	na	na	na	na	NA	2	0	12	0.7	1	0	0	3	65	3	1	1	1	1	2.00	3
1.85	2	2	2	50	1	1	0	1	2	1	1	0	6	1.4	2	2	45	1	1	0	8	1.25	2	0	2	2	45	2	0	0	0	0	0.50	0
1.35	2	0	3	55	0	1	0	1	2	0	1	0	10	1.4	2	2	50	2	2	0	12	1.1	2	1	2	3	55	2	1	1	0	0	5.50	0
1.3	2	0	2	54	0	1	0	1	2	0	2	0	10	1.2	2	2	50	2	1	0	10	1.45	2	0	0	2	45	1	1	1	1	2	4.00	2
1.8	2	2	2	40	1	1	0	1	1	0	1	0	8	1.9	2	2	40	1	1	0	10	1.4	2	0	2	2	40	1	0	0	0	0	4.00	0
1.45	0	2	2	70	0	1	0	1	3	0	2	1	5	1.3	0	2	50	2	2	1	8	1.15	0	0	0	2	55	2	0	0	0	0	6.00	0
1.6	3	0	2	53	1	1	1	1	2	0	1	0	10	1.7	2	2	46	2	1	0	5	1.85	2	0	0	2	35	1	0	0	0	0	9.00	0
1.7	2	0	3	50	1	1	0	1	2	0	1	0	4	1.8	2	3	NA	NA	1	0	4	1.75	3	0	0	3	46	2	0	0	0	0	7.00	0
2.02	0	1	2	44	1	1	0	1	1	0	1	0	10	1.7	1	2	38	1	1	0	12	1.62	1	1	2	1	33	1	0	0	0	0	9.00	0
1.415	2	0	1	47	0	1	0	1	2	0	1	0	4	1.6	2	1	40	1	1	0	6	1.55	2	0	0	2	36	1	0	0	0	0	9.00	0
1.48	2	0	0	NA	0	1	0	1	NA	0	1	0	6	1.3	1	0	NA	NA	1	0	18	1.4	0	0	0	0	NA	NA	0	0	0	0	8.00	0
1.41	1	0	2	80	0	1	0	1	3	0	1	0	10	1.4	1	2	NA	NA	1	0	6	1.4	1	0	0	1	40	2	0	0	0	0	8.00	0
1.6	1	3	0	NA	1	1	0	1	NA	0	1	0	6	1.5	1	1	46	2	2	0	14	1.05	0	2	3	1	60	3	0	0	0	0	6.00	0
1.65	2	2	2	42	1	1	0	1	1	0	1	0	4	1.6	2	0	NA	NA	1	0	6	1.4	2	0	2	0	NA	NA	0	0	0	0	4.00	0
1.6	2	2	2	40	1	1	0	1	1	0	1	0	8	1.6	2	2	25	0	2	0	9	1.2	2	0	2	1	35	1	0	0	0	0	1.00	0
1.4	2	0	0	80	0	1	0	1	3	2	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	0.00	na

Final follow up (maternal+ children)

yr of reinterv	Delivery (1- normal, 2- elective Cesarian, 3-emergency cesarian , 4- still born 5- mtp/abortion, 6- IUD)	birthweight	b. weight grade	FetalComplication (0- none, 1- prematurity, 2- Prolonged ICU stay, 3-resp distress	age	present Weight	P wt grade(0- no malnutrition, 1- grade 1, 2- grade2, 3- grade 3, 4- grade 4)	Present Height	P Ht grade (0- no stunting, 1- grade 1, 2- grade2, 3- grade 3)	Headcircumference	grade(0-normal, 1-microcephaly)	Milestones (1-normal, 2-abnormal)	Abnormalities
0	1	2.8	1	0	4	16	0	103	0	50	0	1	0
0	1	2.75	1	0	9	20	2	125	1	53	0	1	0
0	1	3.3	1	0	0.5	5.5	1	55	1	40	0	1	0
0	1	2	2	0	4	10	2	99	0	45	1	1	0
0	1	2.1	2	0	6	17	1	106	1	51	0	1	0
0	1	2.4	2	0	6	18	0	107	1	50	0	1	0
0	1	2.8	1	0	3	15	0	101	0	48	0	1	0
0	1	2	2	0	9	20	2	118	1	50	0	1	0
0	1	2.5	1	0	5	16	0	109	0	51	0	1	0
0	2	2	2	0	7	15	2	108	2	48	0	1	0
0	2	2.5	1	0	1.5	7	2	72	2	45	0	1	0
0	1	2.45	2	0	0.75	7	1	64	2	44	0	1	0
2002	4	NA	NA	NA	NA	NA	NA	NA	NA	NA		NA	NA
0	1	2.5	1	0	2.5	11	0	88	0	47	0	1	0
0	1	2.5	1	0	6	17	1	110	0	50	0	1	0
0	1	2.75	1	0	4	11.5	1	99	0	50	0	1	0
0	2	2	2	0	2.5	11	0	65	3	43	1	1	0
0	2	2.3	2	0	0.75	8.5	0	67	0	42	0	1	0
0	2	2.55	1	0	4	13	0	80	0	48	0	1	0
0	1	2.75	1	0	5	16	0	104	0	55	0	1	0
2005	1	2.2	2	0	10.5	25	1	126	0	55	0	1	0

0	5	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	2	2.9	1	0	4.5	20	0	108	0	52	0	1	0
0	2	2.4	2	0	8	36	0	132	0	54	0	1	0
0	2	2.5	1	0	3.5	14	0	98	0	50	0	1	0
0	2	3.25	1	0	8	18	1	114	1	47	0	1	0
0	2	3.2	1	0	8.5	35	0	133	0	55	0	1	0
2007	2	2.5	1	0	10	27	0	125	1	56	0	1	0
0	1	1.75	2	1	2	11	0	100	0	45	1	1	0
0	1	2.8	1	0	2	12	0	100	0	50	0	1	0
0	2	2.5	1	0	0.5	6.5	0	65	0	45	0	1	0
0	1	2.25	1	0	4	16	0	110	0	50	0	1	0
0	1	2.5	1	0	6.5	18	0	112	0	56	0	1	0
0	3	3.2	1	0	8	22	0	120	1	56	0	1	0
2010	1	1.8	2	1	9	23	1	121	1	55	0	1	0
0	2	2.35	2	0	5	18	0	110	0	57	0	1	0
0	1	3.5	1	0	4	18	0	110	0	55	0	1	0
0	1	2.75	1	0	5.5	19	0	106	0	54	0	1	0
0	1	2.8	1	0	5	18	0	108	0	55	0	1	0
0	2	2.6	1	0	2.5	12	0	83	12	47	0	1	0
0	1	3	1	0	9	24	1	128	0	65	0	1	0
0	1	2.3	2	0	4.5	16	0	100	1	45	0	1	1
0	2	1.3	3	2	0.75	6	2	65	2	40	1	1	0
0	1	2.6	1	0	2	11	0	90	0	45	0	1	0
0	1	2.85	1	0	1	9	0	75	0	45	0	1	0
0	2	2.4	2	0	1.5	10	0	80	0	45	0	1	0
0	1	2.3	2	0	6	18	0	110	1	55	0	1	0
2010	6	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	1	2.55	1	0	0.75	8.5	0	70	0	42	0	1	0
0	1	2.4	2	0	5.5	19	0	110	0	50	0	1	0
1999, 2009	2	2.2	2	0	10	30	0	125	0	54	0	1	0
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	na	NA	NA
na	na	na	na	na	na	na	na	na	na	na	na	na	na

Report II

LV Dyssynchrony: Quantification by Speckle tracking and Tissue Velocity derived strain indices and comparison between Subjects With or Without Systolic Dysfunction and with or Without Left Bundle-Branch Block

CONTENTS

Introduction

Cardiac resynchronization therapy (CRT) improves symptoms and survival of patients with advanced systolic heart failure; however, up to 30% of patients do not improve after CRT when selected by current recommended criteria. [1, 2] To improve patient selection, many studies have quantified the magnitude of intra-ventricular mechanical dyssynchrony. Systolic velocity timing derived from tissue Doppler imaging has been used most frequently, and several indexes have been reported to predict positive responses to CRT.[3-7] However, the multicenter Predictors of Response to CRT (PROSPECT) study recently reported that no echocardiographic dyssynchrony parameter could be recommended to improve patient selection for CRT beyond current guidelines [8, 9]

Two Dimensional Echocardiographic strain and strain rate imaging is a new technology enabling more reliable and comprehensive assessment of myocardial function. The spectrum of potential clinical applications is very wide due to its ability to differentiate between active and passive movement of myocardial segments, to quantify intra-ventricular dyssynchrony and to evaluate components of myocardial function, such as longitudinal myocardial shortening, that are not visually assessable. [10, 11]Strain and strain rate imaging initially became possible using tissue Doppler. TDI, like all Doppler-derived measurements, has its own limitations. [12, 13]

Non-Doppler 2D-strain imaging derived from speckle tracking is a newer technique which analyzes motion by tracking speckles (natural acoustic markers) in the 2D ultrasonic image. The advantage of this method is that it is angle independent and also has better spatial resolution & measurement reproducibility.[14-21]

Studies have showed that Speckle derived dyssynchrony indices (based on time to peak strain) were better predictive of response to CRT. [22-25] However, there are very few studies comparing these in various sub groups of populations. Moreover, whether the various cutoff values of echocardiographic dyssynchrony indexes associated with left ventricular (LV) reverse remodeling after CRT are specific for the patients with decreased LV systolic function and/or conduction delay is not well known.

Dyssynchrony has two components, mechanical and electrical & patients might have a variable grade of involvement of both the components. Mechanical dyssynchrony is not necessarily due to electrical dyssynchrony but may develop as a consequence of the so-called contractile disparity. [26] In other words, LV walls with different contractile properties may generate different contracting forces, and this may produce a time delay in the peak of segmental contractions.

In this situation, which is not uncommon in the left ventricles of patients with heart failure, if electrical dyssynchrony is not present, there is no clear evidence that CRT is effective in reducing mechanical strain dyssynchrony, while other interventions, such as b-blockers, have been reported to do so. [27]

CRT is mainly an electrical therapy; it uses electrophysiological principles to modify the mechanical activation of the heart. A clear understanding of electrical conduction in failing heart is under clinical research. The improved mechanical function which is the aim of CRT is also determined by the myocardial properties and the electromechanical coupling at the regional level. CRT needs not only electrical re synchronization but also improved and synchronous mechanical activation. Electrical dyssynchrony may or may not be associated with wasted energy of contraction. Such wastage of contraction is necessarily due to late activation of regions of myocardium; greater will be the wasted energy and loss of efficiency if the late activated areas are contracting well. The greater the wasted energy of contraction greater will be the benefits, at least theoretically of CRT. The chance of non response to CRT is hence higher in patients with large scars in the posterior-lateral areas when effective contraction cannot be achieved even with early activation. Such patients may have lesser energy wastage because of lack of strain development in the diseased myocardium.

Strain Delay Index (SDI- 2D) based on 2D speckle tracking , is a more recently described parameter which takes into account regional strain (an indirect measure of regional deformation) and the temporal distribution of peak strain in multiple myocardial segments. It is a measure of wasted energy due to dyssynchronous mechanical activation of left ventricle. This account for both electrical and mechanical components of dyssynchrony and might be a better predictor of response to CRT as evident in small studies.[28]

Other 2D speckle based dyssynchrony indices under research are the speckle radial dyssynchrony index and strain derived dyssynchrony index, which is standard deviation of time to peak strain in the myocardial segments. Recent study comparing TDI derived strain measurement with speckle derived measurements, in patients with or without systolic dysfunction and with or without left bundle-branch block, showed that tissue velocity based indices had a significant overlap between the various groups and couldn't differentiate between groups. [29] Speckle based dyssynchrony index was more specific for dyssynchrony in patients with systolic dysfunction and left bundle-branch block.

Aims & Objectives

1. Characterization of 2D (Speckle) derived myocardial strain parameters of left ventricular systolic function in subgroups of patients with LV dysfunction and LBBB

   a. Strain delay index

   b. Strain derived dyssynchrony index

2. Validation of Dyssynchrony indices based on 2D strain and comparison with TDI based indices in patients with or without systolic dysfunction and with or without left bundle-branch block

Review of Literature

The acute adverse effects of left ventricular (LV) dyssynchrony on cardiac performance were first described in 1925 by Carl Wiggers. [30] Over the past decade, cardiac resynchronization therapy (CRT) has changed the treatment of patients with end-stage heart failure. In recent years, the accurate diagnosis of LV dyssynchrony has become the focus of myriad publications, driven by the advent of cardiac resynchronization therapy (CRT) to treat heart failure due to severe LV dysfunction in the setting of marked prolongation of the QRS interval. [31-33] In the initial large clinical trials of CRT, QRS duration was used as a measure of dyssynchrony to select patients for treatment.[31, 32] However, the sensitivity and specificity of QRS duration to predict response to CRT were less than optimal. [34, 35]

Therefore numerous ''time-to-peak'' parameters based directly on the motion of the LV walls were developed to diagnose LV mechanical dyssynchrony with echocardiography in an attempt to improve CRT selection criteria. [36] Echocardiographic mechanical dyssynchrony parameters initially showed promise in predicting response to CRT in single-center studies. [3-7] However, the multicenter Predictors of Response to CRT (PROSPECT) study recently reported that no echocardiographic dyssynchrony parameter could be recommended to improve patient selection for CRT beyond current guidelines.[8, 9]

In addition, the Resynchronization Therapy in Narrow QRS (RETHINQ) trial recently reported that patients with narrow QRS intervals and evidence of mechanical dyssynchrony do not benefit from CRT.[37]

Currently, on the basis of the guidelines of the American Heart Association, American College of Cardiology, and Heart Rhythm Society, CRT is considered a class I indication in patients with drug-refractory heart failure (New York Heart Association functional class III or ambulatory class IV) with left ventricular (LV) ejection fraction <35%, broad QRS complex (>120 ms), and sinus rhythm. However, when patients are selected according to the aforementioned criteria, approximately 30% do not have beneficial responses on the basis of clinical outcomes or echocardiographic indicators of reverse remodeling, indicating that currently used guidelines are not perfect at identifying patients with heart failure most likely to benefit from CRT. [1,2]

This is a major issue because CRT is a costly procedure and may potentially expose patients to peri-procedural or device-related complications; therefore, it should be reserved for patients with heart failure for whom positive responses can be anticipated, to optimize the allocation of organizational and financial resources and also to avoid unjustified risk exposure. Hence the search for newer methodologies and techniques to assess dyssynchrony and to better predict the response to resynchronization therapy. [2]

Echocardiographic strain and strain rate imaging is a new technology enabling more reliable and comprehensive assessment of myocardial function.

Myocardial deformation

Following electro-mechanical activation, the myocardium deforms during systole due to sarcomere shortening. This active deformation causes a reduction in intracavitary size, resulting in the ejection of blood from the ventricle. In diastole the original ventricular geometry is restored due to active relaxation and passive filling following atrial contraction. Since myocardial tissue is virtually incompressible, the volume of the ventricular wall remains the same during the cardiac cycle and, thus, deforms in three dimensions. During systole there is three-dimensional deformation which can be expressed in three ventricular coordinates: a longitudinal shortening and a circumferential shortening and a radial thickening. [11, 38]

Myocardial deformation is expressed as a one dimensional parameter: strain (e). It defines the total deformation during the cardiac cycle relative to the initial length at the onset of the cardiac cycle, and is expressed in percentages. This implies that longitudinal and circumferential (systolic) shortening result in a negative strain and radial (systolic) thickening in a positive strain.

When the initial length of the investigated myocardial segment is known during the cardiac cycle, the relative length change (strain) can be calculated throughout the entire cardiac cycle. [11, 38]The local end-systolic strain value reflects the regional ejection fraction and the global LV end-systolic strain reflects the LV ejection fraction. When visualized in a graph the different phases of the cardiac cycle can usually be identified: during systole the strain values become more negative (S-wave) with the negative peak at the aortic valve closure, representing the maximal longitudinal myocardial shortening during contraction (or peak systolic strain). In diastole the strain values return towards zero (towards the original length of the analyzed myocardial segment at the onset of the cardiac cycle).[38]

The speed at which the myocardial deformation occurs is the strain-rate (SR) and is expressed in s-1. SR depicts the change in strain over a period of time. Thus, when the myocardium shortens there is a negative SR and, the steeper the slope of the strain-curve, the higher the SR-values. The peak systolic strain rate correlates well to loading independent indices of contractility and hence provides valuable information on regional contractile function. [39, 40] Comparable to strain, when plotted in a graph the different cardiac phases can be recognized.

To optimally assess regional myocardial function, both strain and SR need to be calculated since they provide complementary information: end systolic strain estimates ejection fraction and peak systolic SR is a measure of contractility. By analogy, when driving a car both the total distance of the journey as well as the speed of the car during the journey provide valuable information.

Tissue deformation (myocardial strain) imaging:

Currently there are two different methods to calculate myocardial deformation: tissue Doppler derived strain and two-dimensional strain [10, 41]

As a spatial derivative of velocity, strain rate provides increased spatial resolution for precise localization of diseased segments. However, strain rate needs high temporal resolution (>100 Hz) to avoid underestimation due to under sampling. Therefore, Doppler, because of its high temporal resolution, is superior to speckle tracking for strain rate imaging. However, Doppler-derived strain is angle dependent and highly susceptible to noise arising from the blood pool, aliasing and reverberation. The use of integrated strain helps reduce random noise while maintaining near similar spatial information. The amount of shortening or stretch in the tissue or fibers describes the normal strain, and the amount of distortion associated with the sliding of plane layers over each other describes the shear strain within a deforming body.

There are two methods for assessing deformation on a continuum. One description is made in terms of the material coordinates. This is called ''material description'' or ''Lagrangian description,'' which defines motion around a given point in tissue as it traverses through space and time. Similar to tagged magnetic resonance imaging (MRI), speckle-tracking technology analyzes Lagrangian strain, in which the end-diastolic tissue dimension represents the unstressed, initial material length as a fixed reference throughout the cardiac cycle. [10]

An alternative way to describe deformation is to consider the relative velocity of motion at a particular location in space as a function of time, referencing the region in terms of the spatial coordinates, called the ''spatial description'' or ''Eulerian description.'' DTI analyzes Eulerian strain, which is derived from the temporal integral of the DTI strain rate signal and uses instantaneous lengths for the reference length.[42] In practice, tissue Doppler scanners can convert Eulerian strain into Lagrangian strain. Likewise, by taking the inverse integral of Lagrangian strain, one may also calculate Eulerian strain.[42]

The general state of strain at a point in a body is composed of 3 components of normal strain and 3 components of shear strain. Therefore, for the left ventricle, 3 normal strains (longitudinal, circumferential, and radial)

and 3 shear strains (circumferential-longitudinal, circumferential-radial, and longitudinal-radial) are used to describe left ventricular (LV) deformation in 3 dimensions. One of the principal purposes of LV shearing deformation lies in amplifying the 15% shortening of myocytes into 40% radial LV wall thickening, which in turn results in a >60% change in LV ejection fraction in a normal heart. [43, 44]

Tissue Doppler Imaging (TDI)

Doppler echocardiography is a well-established technique to calculate pressure gradients, quantify valvular stenosis, and measure diastolic function. In 1989, Isaaz et al described the application of Doppler echocardiography for the evaluation of myocardial motion and TDI was born. [45]

Routine Doppler echocardiography employs a low-velocity, high-amplitude filter to remove the artifact produced by the highly reflective blood–tissue interface. When this filtering strategy is reversed, these low-velocity, high-amplitude signals from tissue motion are displayed. Tissue Doppler velocities can be displayed as a spectral trace (pulsed TDI) representing the peak velocities, or as a color map superimposed on the 2D image similar to color Doppler (color TDI).

The advantage of pulsed TDI is that the sample volume can be placed in a small select area, thereby providing data with an excellent temporal resolution. The color TDI has the advantage of displaying velocities over a wide area of myocardium at the same time, thus allowing multiple segments to be analyzed simultaneously, though this comes at the expense of decreasing temporal resolution. As with color Doppler, the velocities from color TDI are mean velocities whereas those from pulsed Doppler and pulsed TDI are peak velocities. [10, 11, 45]

Limitations of Tissue Doppler-derived strain:

TDI is dependent upon the angle of insonation and is only able to estimate strain along the ultrasound beam and thus cannot reliably measure strain in the azimuth or perpendicular plane. If the tissue under investigation is not moving perfectly in line with the ultrasound beam then the measurement obtained will be subject to inaccuracy (dependent on Cos ? of the angle of insonation). This limits the use of TDI-derived-strain measurements primarily to longitudinal fibers with the inability to quantify deformation in the radial plane. [13]

Furthermore in patients with left ventricular dilatation, the full extent of the myocardial wall cannot be aligned within the ultrasound beam. As with all Doppler-derived data, if the angle of interrogation between the tissue motion and ultrasound beams is greater than 20^0, the peak values will be significantly reduced. The second limitation is that the velocities of the tissue along the Doppler beam are in reference to the transducer. TDI is unable to resolve the difference in motion of a myocardial segment that could be actively contracting or merely being displaced due to the tethering effect from the adjacent segment. [13, 46]

Parameters of SR and strain can also be derived from myocardial velocity data. Calculations of strain and SR from TDI data have several pitfalls. First, if the TDI data are poor, the strain/SR measurements will contain errors. For instance, if a myocardial segment suffers from echo dropout, the TDI will encode that segment as having a lower velocity than it may truly have, leading to erroneously low strain and SR and a suggestion of hypokinesia or akinesia. Reverberations and side lobes can also introduce error that may overestimate, underestimate, or simply generate random noise in the strain/SR values.[13, 46]

The second limitation of TDI-derived strain values comes from deviation in the angle of interrogation. Deviation from the intended angle is even more important for strain calculation than it is for TDI velocity data. If one deviates from the true direction of contraction when measuring velocities, the peak velocities will simply be reduced. If one alters the angle for strain measurements, the type of strain being measured will change. [13, 46] Taking into account all the technical aspects mentioned above it is not surprising that TDI-derived strain % SR measurements are not easily reproducible (>10– 15% inter observer variability). This is one of the explanations why this technique has not become standard in daily practice. [12, 47]

SPECKLE TRACKING OR NON-DOPPLER 2D STRAIN IMAGING

General Principles

Non-Doppler 2D strain imaging is a newer technique for obtaining strain and SR measurements. It analyzes motion by tracking speckles (natural acoustic markers) in the ultrasonic image in two dimensions. These markers are statistically equally distributed throughout the myocardium. The size of these elements is 20 to 40 pixels. Each speckle can be identified and followed accurately over a number of consecutive frames. [14-16, 48] These markers, within the ultrasonic image, are tracked by calculating frame to frame changes using a sum of absolute difference algorithm.

The speckles are tracked from one frame to the following one, thereby calculating the velocity field for that frame only. Rotation and motion of the heart in the chest cavity, and breathing, account for most out-of-plane motion. However, these cause the disappearance of the speckles over a few frames, rather than within two consecutive frames. Current available software allows spatial and temporal image processing with recognition and selection of such elements on the ultrasound image. The geometric position of these speckles changes from frame to frame with the surrounding tissue motion. The geometric shift of each speckle represents local tissue movement. By tracking theses speckles, 2D tissue velocity, strain, and SR can be calculated. [14-16, 48]

Technique : [48]

Non-Doppler 2D strain imaging is simple to perform. It requires only one cardiac cycle to be acquired; further processing and interpretation can be done after image data acquisition. Because it is not based on tissue Doppler measurements, images are easier to obtain as they are angle independent–it is not necessary for the main motion vector to be parallel to the beam. The 2D loops from the routine echocardiographic examination are processed offline. The software is dependent on high-resolution image quality and applied with harmonic imaging. Within the end-systolic frames an estimation of the LV myocardium is traced in a click-to-point approach.

Subsequently the software automatically defines an epicardial and mid myocardial line and processes all frames of the loop. Endocardial border is identified by edge detection, based on black-and-white transition recognition on a single frame. The myocardium is defined by empiric estimation of myocardial thickness and can then be further corrected by the operator. By tracking the entire LV myocardium, the new border is determined without the need for repeated border location by edge detection. Motion and velocities are then analyzed by calculating frame-to-frame changes. The final result showing is a continuous cineloop, tracking the acoustic markers and superimposing color points on the gray scale image. A visual control for the individual tracking quality is directly performed thereafter. [14-16, 48]

Tracking quality is based on several criteria:

1. Each speckle is followed for several consecutive frames forward and backward. Return to baseline coordinates is considered evidence for adequate tracking.

2. Adjacent speckles (that are in close proximity within the tissue) are assumed to have similar velocities. If a significant difference in tissue velocities is detected, the tracking is rejected by the software.

3. The software evaluates the drift compensation that is required. The strain at the beginning and the end of a cardiac cycle should be the same. The larger the required drift compensation, the lower the tracking quality score.

The automatically obtained tracking process may be accepted or rejected by the reader. Several adjusting tools are available to optimize the tracking quality. The myocardium in each of the 3 standard apical planes is automatically divided into 6 segments and the analyzed. These diagrams can display different parameters (strain, SR, displacement, velocities), which are all derived from the instantaneous angle-independent speckle velocities. [14-16, 48] For accurate speckle tracking, a high frame rate is important. Speckle patterns change over the course of the cardiac cycle because of deformation of the heart and out-of-plane motion. A high frame rate decrease the speckle change between frames, allowing better tracking. On the other hand, a lateral resolution with high beam density is also important for good speckle tracking. But, higher beam density means lower frame rate. There is no definitive conclusion on how to balance these two parameters, but good clinical results are reported with frame rates of 40–60 frames per second. [14-16, 48-50]

Validation of speckle tracking echocardiography:

Speckle tracking requires a thorough understanding of echocardiographic imaging technique for both image acquisition and myocardial border tracing. In addition, images must be of high-resolution quality to track regions of interest accurately. Myocardial strain derived from STE has been validated using sono-micrometry and tagged MRI and correlate significantly with tissue Doppler–derived measurements. [17-21]

Tissue Doppler technology is dependent on achieving a parallel orientation between the ultrasound beam and the direction of motion and therefore is applied mostly in apical views for recording longitudinal strains and from mid anterior, and mid inferior segments of the left ventricle in short-axis views for recording radial strains. STE, in contrast, can analyze the longitudinal and radial deformation of all LV segments from apical views and radial and circumferential strain of all LV segments from the short axis views. In comparison with DTI, receiver operating characteristic curve analysis has shown that longitudinal and radial strain measured using STE has a significantly greater area under the curve than DTI strain in differentiating normal and dysfunctional segments. [14-16, 48-50]

STE do not require scaling for any index of LV morphology. Overall, speckle tracking appears to be highly reproducible and minimally affected by intra observer and inter observer variability. [17-21]

Suffoletto et al. have shown that 2D-strain-derived values are a good predictor of clinical response to CRT. Speckle-tracking radial strain can quantify dyssynchrony and was a better predictor immediate and long-term response to CRT and has potential for clinical application. They also found a significant difference between those patients who had lead tip pacing at their latest site of activation compared with those who had other segments paced.[22]

Pascal Lim et al have shown that use of strain delay index with longitudinal strain by speckle tracking has a strong predictive value for predicting response to CRT in both ischemic and non-ischemic patients. This can predict response to CRT by directly assessing the potential for incremental contractility gain after resynchronization rather than by simply quantifying LV dyssynchrony by regional timing.[29]

Mele et al in their study reported that standard deviation of the averaged time-to-peak strain (TPS-SD, ms) of 12 middle and basal LV segments obtained from the three standard apical views was predictive of response to CRT. A cut off value of =60 ms for TPS-SD was significantly associated with responder identification.[25]

Chinami Miyazaki, et al compared tissue velocity– derived and strain-derived dyssynchrony indexes in patients with or without systolic dysfunction and left bundle-branch block. A substantial proportion of normal subjects have tissue velocity– derived dyssynchrony indexes higher than the cutoff value proposed for predicting beneficial effect of cardiac resynchronization therapy. In this study almost 100% of patients with systolic dysfunction showed dyssynchrony on using the proposed strain-derived cutoff value in the Mele et al study. The reason for this discrepant result is not clear, but they speculated that a cutoff value that can predict the effect of CRT among heart failure population is higher than the one that can separate a normal from an abnormal population. But overall the study showed that Strain-derived timing index appears to be more specific for dyssynchrony in patients with systolic dysfunction and left bundle-branch block.[28]

Patients & Methods

Subject Population:

A total of 43 patients were examined, and were divided into 4 groups.

1. Group 1 (n=12), left bundle-branch block (LBBB) and LV dysfunction
2. Group 2 (n=11), normal left ventricular ejection fraction & LBBB
3. Group 3 (n=10), left ventricular dysfunction and no LBBB; and
4. Group 4 (n=10), normal subjects.

LBBB was diagnosed when the QRS duration was= 120 ms, with QS or rS complex in lead V1 and a monophasic R wave in leads I and V6. LV dysfunction was diagnosed when ejection fraction was = 35%

Patients with significant non cardiac disease, awaiting or post (< 3 months after) coronary revascularization treatment and severe mitral regurgitation were excluded. Those with LV dysfunction were studied after stabilization optimally with medical treatment. The patients were enrolled after an informed consent

Echocardiography Examination

A comprehensive 2D & Doppler echocardiography was performed with commercially available ultrasound equipment. LV volumes and LVEF were calculated by the biplane Simpson method. Color-coded tissue Doppler imaging was acquired from 3 apical views with IE 33 (Philips Medical Systems).

Images of individual walls with narrow sectors and the shallowest depth were obtained to minimize the angle between the ultrasound beam and the longitudinal axis of the cardiac wall with the highest possible frame rate. Gain settings and pulse repetition frequency were adjusted to optimize color saturation and to avoid aliasing. A single operator acquired images were used to avoid variability and errors. All analyses were performed offline. The data was transferred to Excel sheets and the indices were computed.

TDI Analysis

LV dyssynchrony was defined using the

1. SD in 12 segments of time to peak velocity by TDI (Yu index)
2. Septal-lateral delay of time to peak velocity (TSL)
3. 12 segment maximum delay of time to peak velocity (MD-TDI)

The process can be summarized as follows:

a) Basal and mid ventricular velocity curves (n=12) were derived from color TDI sequences

b) Time to peak velocity was defined when peak velocity reached its maximum positive value during the systolic ejection period.

c) The reference timing point was defined at end diastole (at the peak of the R wave on the ECG tracing), and the systolic period was defined by aortic valve opening and closure with LV outflow tract pulse-wave Doppler flow.

d) Segments with only negative velocity during the systolic ejection period were excluded from the calculation of SD-TDI and TSL.

Significant LV dyssynchrony was considered when Yu index =33 ms, TSL was >65 ms and MD-TDI =100ms as per the present recommendations.

2D Strain analysis: 2D Strain imaging was performed as described earlier. [48]

a) Image acquisition. Images were acquired at a frame rate of 40–60 frames per second with optimal gain and window settings to include the area of interest. A loop of four cardiac cycles were archived for each of the three views – Apical four chamber, two chamber and three chamber views. Breath holding for the period of acquisition was used to reduce relative tissue motion in all cases.

b) Strain (**e**) waveforms were generated using the QLAB software. The strain is calculated as a time integral of strain rate in which the integration starting point was adjusted to the onset of the QRS.

c) The timing of aortic valve opening and closing were estimated based on the time of aortic flow from the pulsed-wave Doppler signal in the apical long-axis view

d) The timing of mitral valve opening and closing was estimated based on the time of mitral flow from pulsed wave Doppler signal in apical four chamber view.

e) The timing thus acquired were superimposed on the waveform analysis

The following indices were calculated:

1. Strain derived dyssynchrony index: Time to peak longitudinal strain (**e**) by speckle tracking was used to calculate the 16-segment SD of time to peak- **e**. In segments with positive or biphasic strain curve, time to absolute maximal **e** was chosen to compute standard deviation
2. Strain Delay Index: In dyssynchronous ventricles, delayed segments do not contribute fully to end-systolic (ES) function. The wasted energy per segment caused by dyssynchrony can be expressed mathematically as the difference between peak ($\mathbf{e}_{peak}$) and ES strain ($\mathbf{e}_{ES}$). Theoretically, this difference ($\mathbf{e}_{ES}$- $\mathbf{e}_{peak}$) increases with the severity of dyssynchrony. The wasted energy is expected to be greater in the segment with preserved contractility than in the segment with no or minimal residual contractility at a similar degree of delayed contraction. This can be well appreciated in scar or fibrotic myocardium.

Methods:

a) The QLAB software generated the strain curves using the acquired echo loops. Before generating the curves, the myocardial borders were manually adjusted and gain settings optimized. Accurate tracking of the speckles were confirmed by observing the movement vectors and analyzing the displacement and strain curves. Tracking zones were manually adjusted to get the best results.

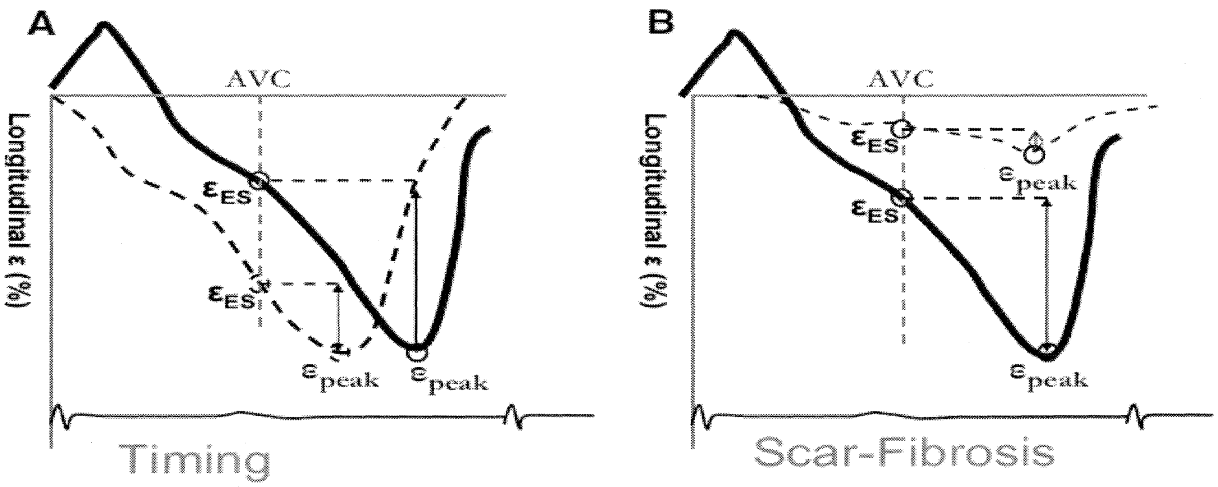
b) The time to the peak of the global **e** curve was used to determine the timing of ES and to compute the $\mathbf{e}_{ES}$ in the 16 segments.

c) Peak and time to peak **e** in the 16 segments were calculated at the point where the **e** curve reached its minimum value during the cardiac cycle.

d) The difference ($\mathbf{e}_{ES}$- $\mathbf{e}_{peak}$) in each segment (all 16 segments) was summed to generate the strain delay index.

e) For a segment that exhibited positive strain or biphasic strain with a peak positive **e** greater than the maximal absolute negative strain, the term ($\mathbf{e}_{ES}$- $\mathbf{e}_{peak}$)) was entered as zero for the calculation of strain delay index.

Figure1: Strain delay index



$$\text{strain delay index} = \sum_{1}^{n} (\varepsilon_{peak} - \varepsilon_{ES})$$

Statistical analysis:

The data were expressed as mean ± SD for normally distributed continuous variables and as absolute frequencies and relative percentages for categorical variables. Normally distributed continuous variables were compared by means of 1-way ANOVA for overall comparison and the Scheffe multiple comparison test for post hoc multiple pair-wise comparisons. The Chi square test was used to compare categorical variables. Dyssynchrony indexes were summarized with the median and 25th and 75th percentiles because they were not normally distributed. Dyssynchrony indexes were compared by the Kruskal-Wallis test for overall comparison. For multiple comparisons among 4 groups (6 pair-wise comparisons), the Mann-Whitney test was used for each pair of groups, and correction was applied for the adjustment of significance level. Values of $P\_0.05$ were considered statistically significant. Multiple linear regressions on the natural logarithm–transformed dyssynchrony indexes were performed to assess the associations with disease group after adjustment for age and gender.

Observations & Results

Characterization of groups:

Group 1: LBBB with LV dysfunction

Group 2: LBBB with Normal LV function

Group 3: No LBBB with LV dysfunction

Group 4: No LBBB with normal LV function (Normal)

Table 1: Distribution of demographic variables

		Group 1	Group 2	Group 3	Group4	P
Number		12	11	10	10	
Age (Yr)		61.1 ± 10.4	58.5 ± 11.4	46.1 ± 13.8	38.9 ± 9.1	<0.001
Male Sex, n(%)		9 (75)	5 (45.5)	8 (80)	9 (90)	0.120
NYHA FC III-IV n(%)		8 (66.7)	0 (0)	6 (60)	0 (0)	<0.001
H/O	H/o CCF	4 (33.3)	0 (0)	5 (50)	0 (0)	0.223
	ACS	5 (41.7)	2 (18.2)	1 (10)	0 (0)	0.438
	Interventions	2 (33.3)	1 (9.1)	1 (10)	0 (0)	0.449
Risk factors	Diabetes	9 (75)	3 (27.3)	3 (30)	3 (30)	0.057
	Hypertension	7 (58.3)	6 (54.5)	3 (30)	3 (30)	0.379
	Smoking	5 (41.7)	3 (27.3)	5 (50)	4 (40)	0.759
	Dyslipidemia	11 (91.7)	11 (100)	7 (70)	8 (80)	0.208
	Family History	4 (33.3)	3 (27.3)	1 (10)	4 (40)	0.476
Segments excluded		14/192(7.3%)	10/176(5.7%)	14/160(8.75%)	8/160(5%)	0.105

Table 2: Distribution of echocardiographic variables.

		Group 1	Group 2	Group 3	Group4	P
QRSD(ms)		148.3 ± 14	146.4 ± 10.3	98 ± 9.2	88 ± 7.9	<0.001
LVIDD		59.7 ± 7.2	50.9 ± 6.8	63.9 ± 6.1	45.1 ± 4.1	0.001
LVISD		48.6 ± 7.2	31.7 ± 5.5	51.7 ± 10.7	26.3 ± 1.8	<0.001
EDV (ml)		155.8 ± 34.9	85.5 ± 12.2	145.2 ± 26.2	81.5 ± 10.1	<0.001
ESV (ml)		107.4 ± 27.1	35.4 ± 8.1	107.7 ± 24.3	31.3 ± 4.3	<0.001
EF (%)		28.2 ± 6.4	59.4 ± 8.4	27.1 ± 7.3	63.8 ± 5.9	<0.001
LA size		41.64±4.34	36.8±1.75	40.20±3.49	31.80±1.92	<0.001
MR	Mild, n (%)	9 (75)	8 (9)	5 (50)	6 (60)	0.074
	Moderate	3 (25)	0 (0)	4 (40)	0 (0)	
PAMP(mm of Hg)		28±11.09	20.36±1.97	30.40±14.28	18.4±1.26	<0.001

Patient demographic characteristics are summarized in table 1, and the echocardiographic data in table 2. The mean age of group 4 was significantly younger than that of the other 3 groups. Females were relatively more common in group 2 (LBBB with normal LV function). Previous history of ACS and diabetes were significantly more common in group 1 than the other. Other baseline values were as expected from selection of the patients based on LVEF and LBBB. Mean QRSD was not significantly different in the groups with LBBB ± LV dysfunction (148.3 ± 14 ms in group 1 vs 146.4 ± 10.3 in group 2). LV dimensions, volumes and Ejection fractions were comparable in both the groups with LV dysfunction.

Table 3: Comparison of Dyssynchrony Indexes Among the 4 Groups*

	Group 1	Group 2	Group 3	Group4	P Value[?]	
S-L walldelay (ms)	95 (58.5, 106.5)	74(69.5, 76)	55 (31, 68)	38 (27, 55)	Total	0.003
					1 vs 2	0.316
					1 vs 3	0.006
					1 vs 4	<0.001
					2 vs 3	0.075
					2 vs 4	0.012
					3 vs 4	0.393
12 segment MaxTDI delay (ms)	139.5 (114.5, 164)	107 (99, 132)	98.50 (74, 135)	65 (50, 75)	Total	0.003
					1vs2	0.016
					1vs3	0.025
					1vs4	<0.001
					2vs3	0.468
					2vs4	0.001
					3vs4	0.011
YU INDEX(ms)	47.95 (36.89, 56.8)	39.41 (36.76, 42.62)	34.09 (25.85, 42.24)	27.66 (16.65, 28.63)	Total	0.003
					1vs2	0.104
					1vs3	0.021
					1vs4	<0.001
					2vs3	0.314
					2vs4	<0.001
					3vs4	0.035
Strain dyssynchrony index (ms)	71.33 (64.47,85.88)	53.76 (47.55, 57.73)	48.46 (40.67, 54.30)	35.87 (33.28, 39.64)	Total	<0.001
					1vs2	<0.001
					1vs3	<0.001
					1vs4	<0.001
					2vs3	0.900
					2vs4	0.025
					3vs4	0.004
Strain delay index (ms)	28.65 (22.66, 30.95)	22, (15.24, 23)	18.6 (16.32, 24.2)	8.21 (7.2, 8.95)	Total	<0.001
					1vs2	0.004
					1vs3	0.004
					1vs4	0.000
					2vs3	0.915
					2vs4	<0.001
					3vs4	<0.001

*Data are expressed as the median (first, third quartiles), Statistics (For Total - Kruskal-Wallis Test, for between group - Mann-Whitney U Test). P[?] - adjusted for age and sex

Comparison of intra-ventricular dyssynchrony indexes among groups

Comparisons of each dyssynchrony index among groups are shown in Table 3 and Figure 2. All dyssynchrony indexes differed significantly among groups in overall test ($P<0.05$). The SD in time to peak systolic velocity in the 12 left ventricular segments (YU index) was greater in group 1 (median 47.25 ms; 25th and 75th percentiles , 36.89 and 56.8 ms) than group 4 (27.66 ms; 16.65 & 28.63 ms), but there was a considerable overlap of all tissue velocity–derived indexes among 4 groups. Therefore it failed to demonstrate a significant difference in any pair-wise comparisons (especially between group 1 & 2).

Similarly S-L wall delay also had significant overlap between different groups and couldn't reliably differentiate between groups (especially between 1&2, 2&3). The results did not differ much after adjusting for age and sex in both the indices. 12 segment maximum delay was significantly larger in groups 1 (139.5 ms; 114.5 &164 ms), 2 (107 ms; 99 & 132 ms), and 3 (98.50 ms; 74 & 135 ms) compared with group 1 (65 ms; 50 & 75). It provided a better separation of groups, yet no significant difference was found between groups 3 and 4 ($P\_0.60$).

In contrast to the considerable overlap in tissue velocity– derived indexes, a stepwise increase was found with both speckle derived indices in the presence of systolic dysfunction and LBBB.

Speckle derived dyssynchrony index was significantly more in group1 71.33 (64.47 & 85.88) than in other groups [group 2 (53.76; 47.55 & 57.73), group 3 (48.46; 40.67 & 54.30)), group 4 (35.87; 33.28 & 39.64)]. Strain delay index similarly was also significantly more in group1 28.65 (22.66, 30.95) than in other groups [group 2 (22; 15.2 &, 23), group 3 (18.6; 16.32 & 24.2), group 4 (8.21; 7.2 & 8.95)].

Both the indices could differentiate LBBB with LV dysfunction (group 1) with other groups. Except for the slight overlap of values between group 2 and 3 due to which they couldn't well differentiate those 2 groups, they could reliable differentiate tall other groups. The results continued to be significant even after adjustment for age and sex.

Predictive value of speckle derived dyssynchrony index:

Even though strain derived dyssynchrony index has been found to be a better indicator for differentiating normal from abnormal populations more distinctively. No definite cut off has been proposed yet. So in our study, we used strain delay as standard, and derived an arbitrary cutoff value for strain derived dyssynchrony index using ROC curve. A cutoff value of 58.6 was obtained, which had a sensitivity of 82% and specificity of 78%. ROC curve is illustrated in Figure 2

Prevalence of Dyssynchrony Based on Cutoff Value:

Based on available evidence and as per recommendations, 33ms, 65 ms and 100 ms were taken as cut off for YU index, S-L wall delay and 12 segment maximum delay index. Similarly for strain delay index 25% was taken as cutoff based on previous studies. As there was no predetermined cut off for speckle derived dyssynchrony index arbitrarily 58.6 ms was taken as cut off based on ROC curve.

The prevalence of intra-ventricular dyssynchrony based on the cutoff is illustrated in Table 5, 6 and figure 4. Significantly 90% of patients in group 2 (LBBB, Normal LV Function) and 50% in group3 had dyssynchrony based on YU index. Similarly based on the S-L wall delay and 12 segment max delay significant number of patients in group2 and group3 had dyssynchrony (81.8 & 30%, 72.7% & 50 % respectively in both groups).

On the contrary both the speckle derived indices could well differentiate the Group1 for other groups. By Speckle dyssynchrony index, only 27% in group 2 and 10% in group 3 had dyssynchrony. Similarly by strain delay index, only 18% in group 2 and 10% in group 3 had dyssynchrony. None of the group 4 patients had dyssynchrony by any of the parameters

Table 4: Predictive power

Predictive power of Speckle dyssynchrony index using Strain delay index >25 as standard		
Positive if >=	Sensitivity	Specificity
26.0	1.00	0.00
31.2	1.00	0.06
33.0	0.91	0.06
36.0	0.91	0.19
42.3	0.91	0.38
51.3	0.82	0.56
54.5	0.82	0.69
56.5	0.82	0.75
58.6	0.82	0.78
60.7	0.73	0.78
62.0	0.64	0.78
62.8	0.64	0.81
63.6	0.64	0.84
64.5	0.55	0.84
66.4	0.45	0.84
67.6	0.45	0.88
68.6	0.45	0.91
69.7	0.45	0.94
74.8	0.36	0.97
80.5	0.36	1.00
88.0	0.18	1.00
121.3	0.00	1.00

Figure 2: ROC curve

ROC curve for predicting Speckle dyssynchrony index using Strain delay index >25 as gold standard

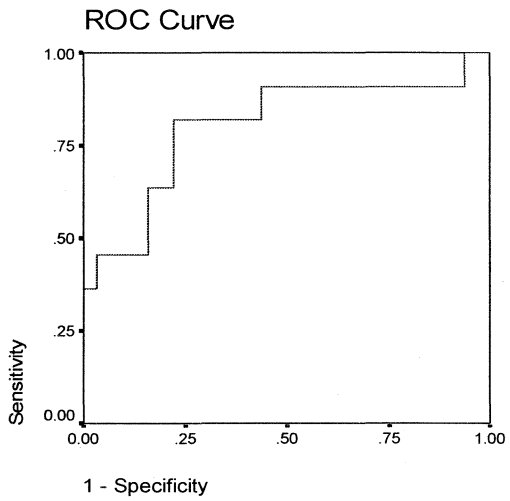


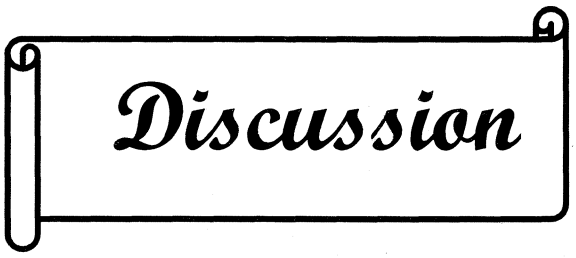
Table 5: Distribution of sample based on tissue velocity based indices.

		Group 1	Group 2	Group 3	Group 4	p value	
YU Index	<33	0 (0)	1 (9.1)	5 (50)	10 (100)	Total	0.000
						1 vs 2	0.296
						1 vs 3	0.006
	>=33	12 (100)	10 (90.9)	5 (50)	0 (0)	1 vs 4	0.000
						2 vs 3	0.043
						2 vs 4	0.000
						3 vs 4	0.012
S L Wall delay	<65	4 (33.3)	2 (18.2)	7 (70)	9 (90)	Total	0.003
						1 vs 2	0.419
						1 vs 3	0.094
	>=65	8 (66.7)	9 (81.8)	3 (30)	1 (10)	1 vs 4	0.009
						2 vs 3	0.019
						2 vs 4	0.001
						3 vs 4	0.276
12 Site Max Delay	<100	1 (8.3)	3 (27.3)	5 (50)	10 (100)	Total	0.000
						1 vs 2	0.242
						1 vs 3	0.033
	>=100	11 (91.7)	8 (72.7)	5 (50)	0 (0)	1 vs 4	0.000
						2 vs 3	0.296
						2 vs 4	0.001
						3 vs 4	0.012

Table 6: Distribution of sample based on speckle based dyssynchrony indices

		Group 1	Group 2	Group 3	Group 4	p value	
Speckle dyssynchrony index	<58.6	0 (0)	8 (72.7)	9(90)	10 (100)	Total	0.000
						1 vs 2	0.000
						1 vs 3	0.000
	>=58.6	12 (100)	3 (27.3)	1 (10)	0 (0)	1 vs 4	0.000
						2 vs 3	0.326
						2 vs 4	0.082
						3 vs 4	0.317
Strain delay index	<25	4 (33.3)	9 (81.8)	9 (90)	10 (100)	Total	0.001
						1 vs 2	0.022
						1 vs 3	0.009
	>=25	8 (66.7)	2 (18.2)	1 (10)	0 (0)	1 vs 4	0.002
						2 vs 3	0.602
						2 vs 4	0.167
						3 vs 4	0.317

For Total - Kruskal-Wallis Test, for pair - Mann-Whitney U Test


Discussion

This study was done with aim of characterising 2D (Speckle) derived myocardial strain parameters (Strain delay index, Strain derived dyssynchrony index) and comparing them with TDI based indices (Septal-lateral delay of time to peak velocity (TSL), 12 segment maximum delay of time to peak velocity (MD-TDI) & Yu index), in 4 groups of patients (grouped on basis of presence or absence systolic dysfunction and/or left bundle-branch block)

Tissue velocity based dyssynchrony indices

Tissue velocity based dyssynchrony indices even though differentiated LBBB with LV dysfunction from normal controls, they showed a significant overlap among different population. Both when used as a continuous variable and as well as using the proposed cut off values, both Yu index and S-L delay index did not differentiate between the major groups. Even though 12 site maximum delay when used as a continuous variable was marginally better, but when used on basis of proposed cut off, it couldn't reliably differentiate between groups 1, 2 & 3. The results are similar to previous study by Miyazaki et al.[28] In a study by Miyazaki et al, which compared similar groups showed that there median value in normal controls was 44, which was significantly more than the cut off value.[28] Contrarily in this study the median value for Yu index on controls is 27.66 (25th and 75th percentiles, 16.65, & 28.63), which is lower than the proposed cutoff value and not quite different from various other studies. Yu et al reported that Yu index was 17.0±7.8 ms in

88 healthy volunteers. Lafitte et al reported a similar value for normal control subject (16±9 ms).[51]

The probable reasons for tissue velocity based indices not being able to differentiate between groups should be considered here. The suboptimal accuracy may be explained by a drawback of the Doppler technique in HF patients in which the signal-to-noise ratio of TDI is particularly affected by myocardial dysfunction (minimal base to apex motion), translational and tethering effects, and mal-alignment of the Doppler sample volume, which increases as LV geometry changes to a globular shape. These issues may explain the difficulty in identifying peak contraction in the flat velocity contour of the failing heart. The inter and intra observer variability is also slightly more than that with speckle derived indices due to the technical issues, and the smaller sample size might also have affected the results.

Speckle derived dyssynchrony indices

Lim et al in their study where they followed up CRT patients reported that speckle derived longitudinal strain delay index appears to be a much more accurate predictor of CRT response.[29] With a proposed cut off value of 25%, it correlated closely with reverse remodeling after CRT in both ischemic and non-ischemic patient populations (r = -0.69, P_0.0001). Importantly, the strain delay index had similar accuracy in patients with ischemic and non-ischemic cardiomyopathies.

This may be explained by the robustness of the index, which considers all 16 myocardial segments of the ventricle, and the fact that the strain delay index is not a simple measurement of contractility or time delay but a combination (and relative weighting) of both of these parameters. Indeed, the delayed myocardial segments incrementally impact the strain delay index value not only in proportion to the severity of dyssynchrony but also relative to the amplitude of their residual contractility. The Speckle derived strain delayed index both when used as a continuous variable and as well as using the proposed cut off from previous studies could well differentiate between the LBBB with LV dysfunction from all other groups comprehensively. There was however some overlap between the group 2 and group3, this could be due to the small sample size.

The tissue velocity indices might truly reflect only the electrical component of dyssynchrony (QRS duration & LBBB), that could be the reason for their inability to differentiate the LBBB with LV dysfunction group from LBBB with normal LV function. This might be the reason why, studies have not showed any further advantage of these indices over the present recognized criteria (QRS duration>120ms). Because QRS duration is a more objective finding it is likely to be more accurate- TDI derived measures are likely to be subjected to more intra observer and inter observer variation.

TDI measures are not validated for general use in the hands of less experienced echo cardiographers. All these may contribute to lack of specificity of the currently used echocardiography criteria.On the contrary Strain delay index which considers mechanical aspects in additional to the electrical dyssynchrony was able to distinguish between the 2 groups more effectively. Also this is evident in recent studies, where it could provide additional information (increasing the predictability of response to CRT) beyond that predicted by present criteria (especially QRS duration).

Using the cut off criteria, 33% of patients in the LBBB LV dysfunction group had a delay index <25% in this study. This might reflect the 30% population of patients, who did not improve after CRT when selected by current recommended criteria. [1, 2] Also it was seen that most of the patients with LV dysfunction and no LBBB, did not have significant dyssynchrony by strain delay index criteria (10% showed significant dyssynchrony). This probably highlights the ability of strain delay index to better identify the patient s who would respond to CRT from groups with LV dysfunction and with or without LBBB. Large randomized studies, in this regard would be needed to come to a definite conclusion.

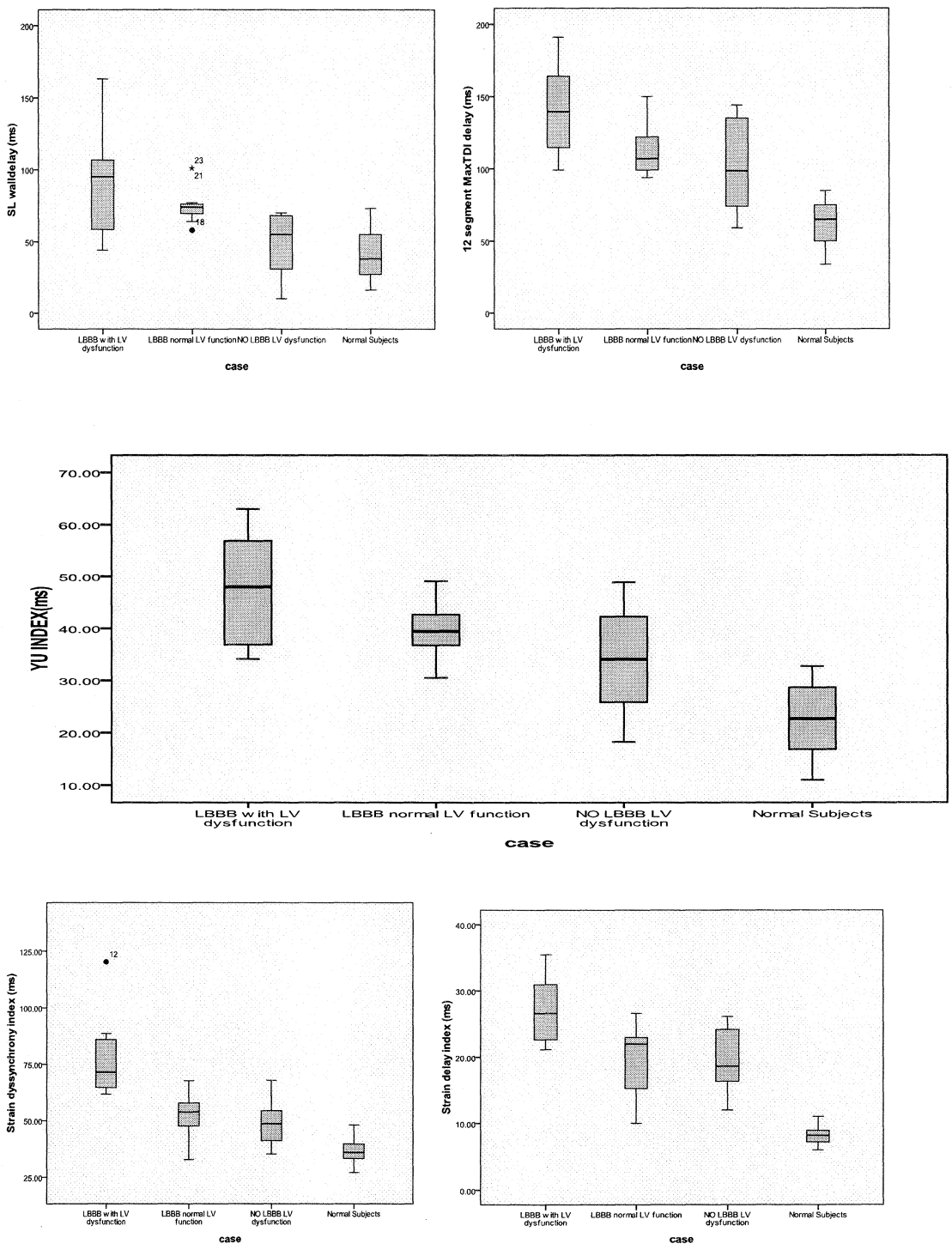


Figure 3. Comparison of tissue velocity– and strain-derived dyssynchrony indexes among groups. The ends of the boxes are the 25th and 75th percentiles; whiskers indicate the minimum and maximum values, excluding outliers

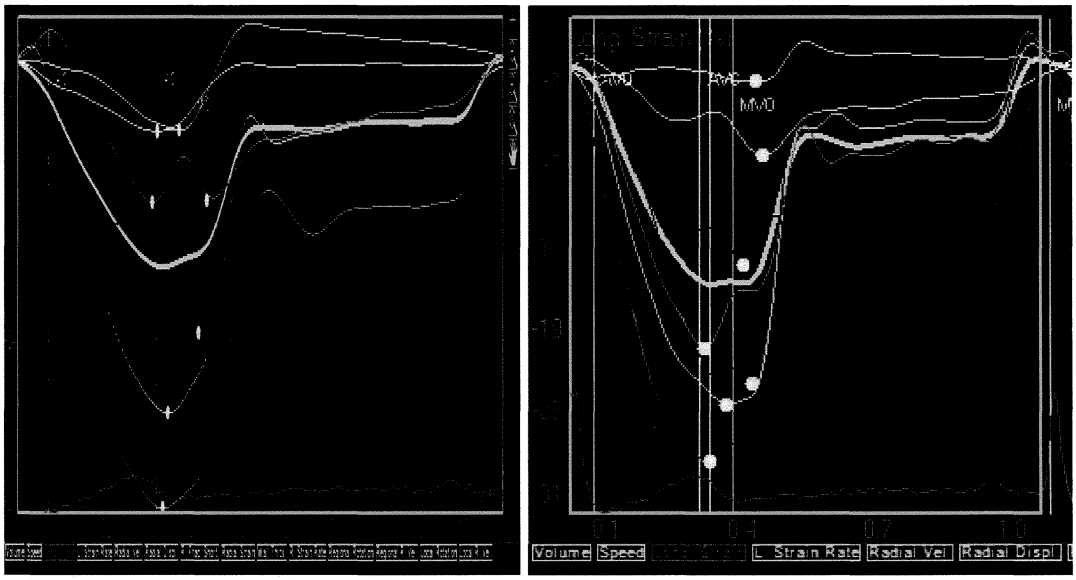


Figure 4: 2D-Strain imaging in control (normal LV function and no LBBB)
Apical 3 chamber and 4 chamber view
Normal peak strain. No dyssynchrony.

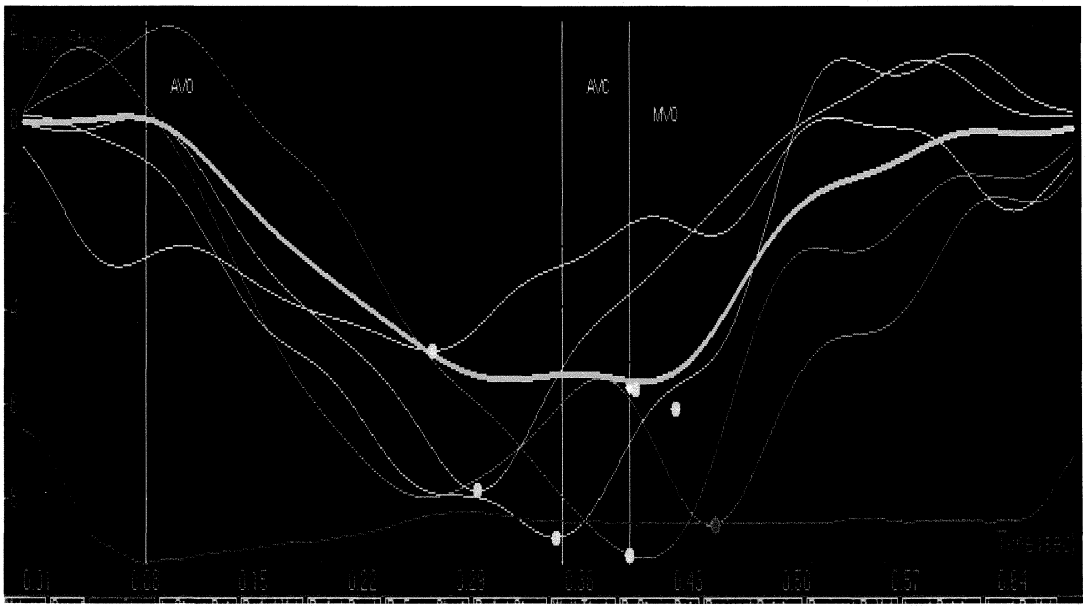


Figure 5: 2D-Strain imaging in Group 1 (LBBB with LV systolic dysfunction)
Apical 4 chamber view
Reduced peak strain with delayed peaks in lateral segments
Significant dyssynchrony present.

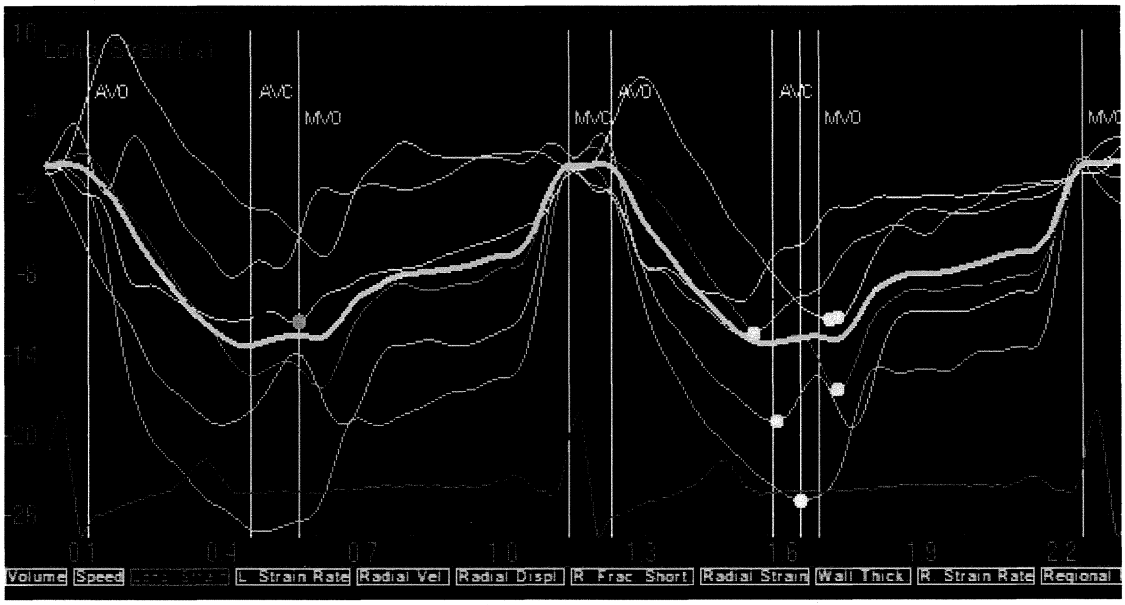


Figure 6: 2D-Strain imaging in Group 2 (LBBB with normal LV systolic function)

Apical 4 chamber view

Normal preserved peak strain, with delay in peak of lateral segments, no significant dyssynchrony.

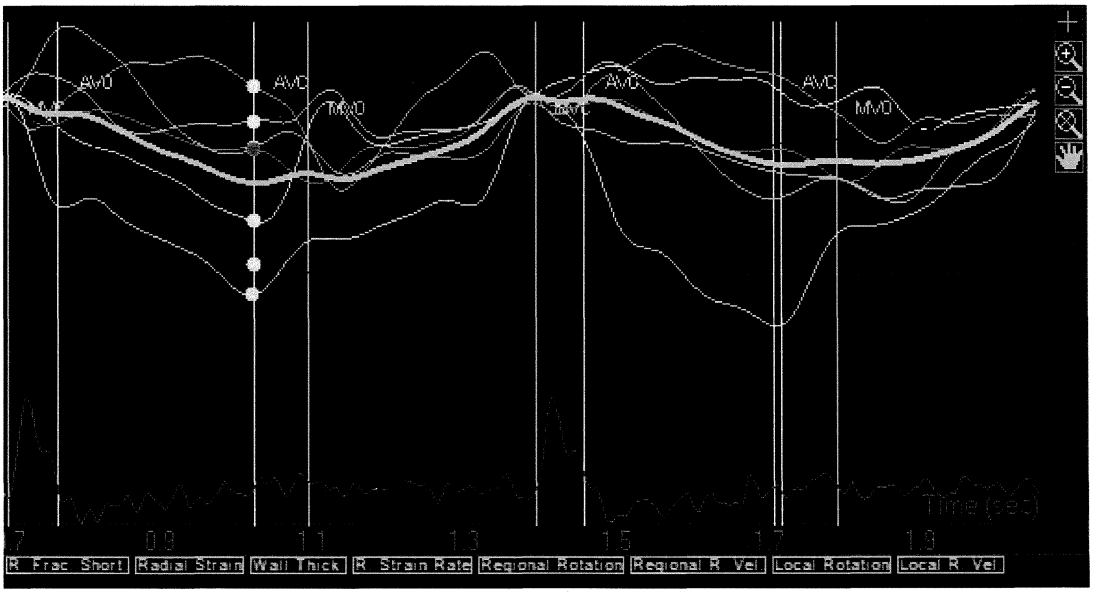


Figure 7: 2D-Strain imaging in Group 3 (no LBBB with LV systolic dysfunction)

Apical 4 chamber view

Reduced peak strain

No significant dyssynchrony.

Similarly Longitudinal strain dyssynchrony index was also able to differentiate the LBBB with LV dysfunction group from the rest similar to previous study done by Miyazaki et al. Unlike the study by Miyazaki et al, where they used 12 segments only, we used 16 segments to calculate the index. As there were no specific predetermined cut off points, we used strain delay index (in v/o its robustness in previous studies, and lack of a proper gold standard) to achieve a arbitrary cut off point of 58.6 in our study. This was nearer to the cut off value of 60ms considered in previous studies using the 12segment model.[25, 28] Using this arbitrary cut off point too, the strain dyssynchrony index was also superior to the tissue velocity indices in differentiating between groups.

Peak strain and systolic tissue velocity represent different mechanical events: Strain peak indicates the end of shortening or the crossover point of myocardial shortening to lengthening, and tissue velocity peak indicates the timing of maximal speed of myocardial motion. There are 2 possible explanations for the superiority of strain to tissue velocity.

First, strain represents regional contraction more reliably because deformation measurements are not affected by tethering and translational motion. Timing of myocardial motion and displacement may underestimate the degree of timing difference in regional contraction compared with deformation, especially if there is more regional heterogeneity in the timing of contraction.

Second, measuring mechanical timing only in the ejection period may underestimate the severity of mechanical dyssynchrony in LBBB patients because the typical mechanical abnormality of LBBB can be observed during the isovolumic periods. Mechanical dyssynchrony in LBBB or the paced heart is characterized by early septal contraction and pre stretching in the lateral wall, which often occurs during the isovolumic contraction period, and post systolic contraction. Therefore, assessment of the isovolumic contraction period and/or post systolic period may have improved the distinction between normal and abnormal groups by speckle derived strain from tissue velocity based indices (where the peaks in isovolumic times are excluded).

Dyssynchrony includes both the electrical and mechanical components. Most of the patients would have a variable proportion of both the components. Patients with LBBB and LV dysfunction may have significant contribution from both the components. Patients in group 2 and group 3 would tend to have only one of them. Normal controls are expected to have neither of them.

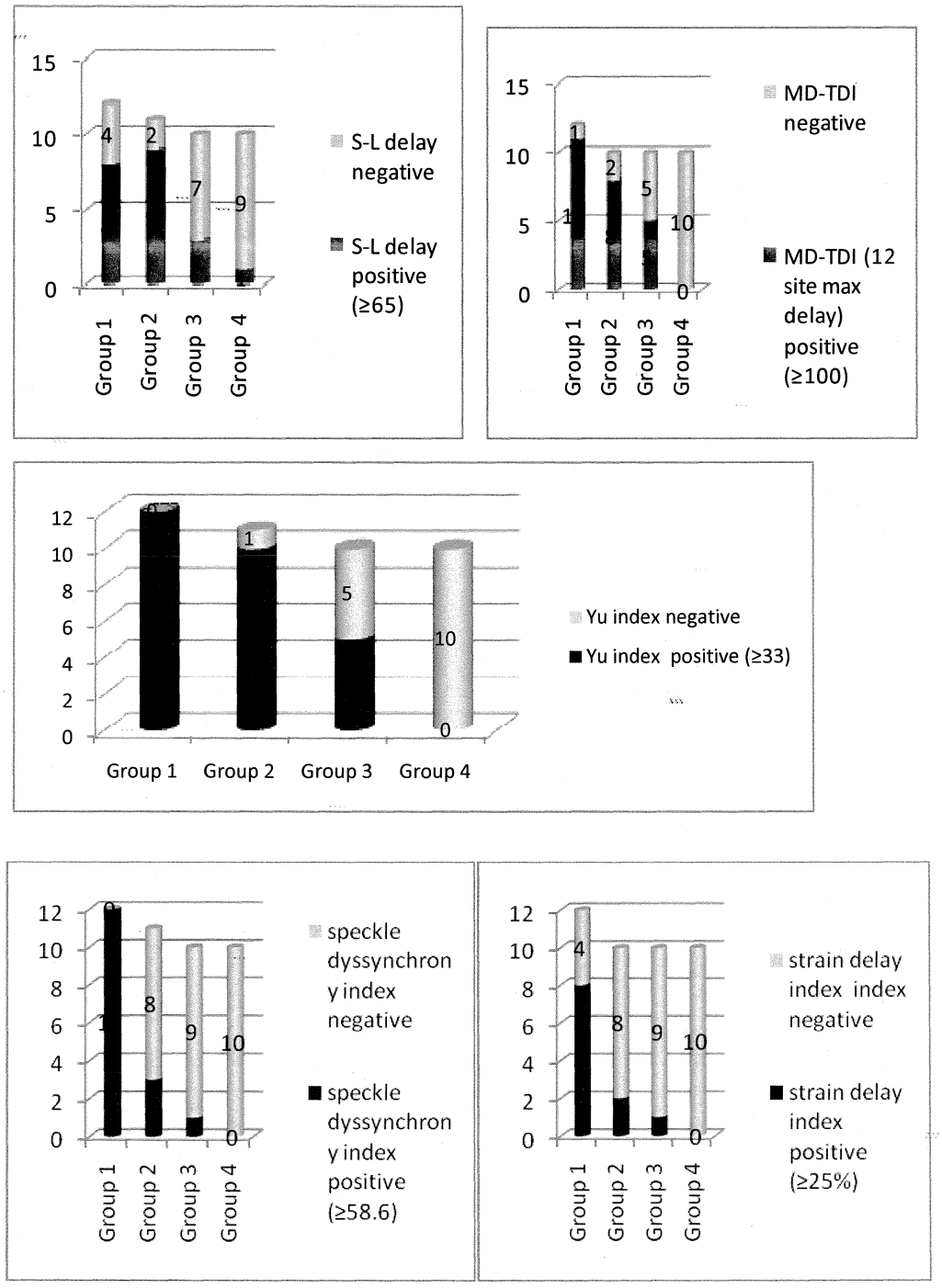


Figure 8: Prevalence of positive findings in each group using different criteria for tissue velocity– derived dyssynchrony indexes. See text for a definition of groups.

This study shows the ability of speckle derived indices, to differentiate the groups significantly due to their robustness in ability to assess the both components of dyssynchrony. The strain-derived dyssynchrony indices distinguished patients with LBBB or decreased LVEF (or both) from those with normal systolic function and normal QRS duration with minimal overlap and appears to identify patients with intra-ventricular dyssynchrony more reliably.

Study Limitations

Study was limited by its small sample size. Also none of the patients underwent CRT placement, so a follow up data on response and clinical correlations are not available. There are no definite objective parameters to assess whether the ability to distinguish LBBB with LV dysfunction from other groups would translate into clinical relevance. Only longitudinal dyssynchrony was assessed in this study, radial and circumferential dyssynchrony were not assessed.

Conclusion

1. There is a substantial proportion of overlap with tissue velocity–derived dyssynchrony indexes among the different population subgroups.
2. 2D speckle Strain-derived dyssynchrony indices are more specific for identifying dyssynchrony in patients with systolic dysfunction and left bundle-branch block.
3. Larger trials comparing the various indices, using responders to CRT as criteria would be needed to identify better predictors of response to CRT.

Bibliography

1. Epstein AE, DiMarco JP, Ellenbogen KA, Estes NAM, Freedman RA, Gettes LS, et al. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities. Circulation 2008;117:e350-408.
2. Dickstein K, Bogale N, Cleland JG, Gitt A, et al. The European cardiac resynchronization therapy survey. Eur Heart J 2009;30: 2450-60.
3. Pitzalis MV, Iacoviello M, Romito R, Massari F, Rizzon B, Luzzi G, et al. Cardiac resynchronization therapy tailored by echocardiographic evaluation of ventricular asynchrony. J Am Coll Cardiol 2002;40:1615-22.
4. Bax JJ, Marwick TH et al. Left ventricular dyssynchrony predicts benefit of cardiac resynchronization therapy in patients with end-stage heart failure before pacemaker implantation. Am J Cardiol 2003;92:1238-40.
5. Bax JJ, Bleeker GB, Marwick TH, Molhoek SG, Boersma E, Steendijk P, et al. Left ventricular dyssynchrony predicts response and prognosis after cardiac resynchronization therapy. J Am Coll Cardiol 2004;44:1834-40.
6. Notabartolo D, Merlino JD, Smith AL, DeLurgio DB, Vera FV, Easley KA, et al. Usefulness of the peak velocity difference by tissue Doppler imaging technique as an effective predictor of response to cardiac resynchronization therapy. Am J Cardiol 2004;94:817-20.
7. Yu CM, Chau E, Sanderson JE, Fan K, Tang MO, Fung WH, et al. Tissue Doppler echocardiographic evidence of reverse remodeling and improved synchronicity by simultaneously delaying regional contraction after biventricular pacing therapy in heart failure. Circulation 2002;105:438-45.
8. Chung E, Leon AR, Tavazzi L, Merlino J, et al. Results of the Predictors of Response to CRT (PROSPECT) trial. Circulation 2008;117:2608-16.
9. Bax JJ, Gorcsan J III. Echocardiography and noninvasive imaging in cardiac resynchronization therapy. Results of the PROSPECT (Predictors of Response to Cardiac Resynchronization Therapy) study in perspective.J Am Coll Cardiol 2009;53:1933-43.
10. D'Hooge J, Heimdal A, Jamal F, Bijnens B, Rademakers F, et al. Regional strain and strain rate measurements by cardiac ultrasound: principles, implementation and limitations. Eur J Echocardiogr 2000;1: 154-70.

11. Gilman G, Khandheria BK, Hagen ME, et al. Strain rate and strain: s step-by-step approach to image and data acquisition. J Am Soc Echocardiogr 2004;17:1011-20.
12. Edvardsen T, Gerber BL, Garot J, Bluemke DA, Lima JA, Smiseth OA. Quantitative assessment of intrinsic regional myocardial deformation by Doppler strain rate echocardiography in humans: validation against three-dimensional tagged magnetic resonance imaging. Circulation 2002;106:50-6.
13. Castro PL, Greenberg NL, GarciaMJ, Thomas JD. Potential pitfalls of strain rate imaging: angle dependency. Biomed Sci Instrum 2000;36:197-202.
14. Reisner SA, Lysyansky P et al. Global longitudinal strain: a novel index of left ventricular systolic function. J Am Soc Echocardiogr 2004;17:630-3.
15. Leitman M, Lysyansky P, Binenbaum M, et al. Two-dimensional strain-a novel software for real-time quantitative echocardiographic assessment of myocardial function. JAm Soc Echocardiogr 2004;17:1021-9.
16. Kaluzynski K, Chen X, Emelianov SY, Skovoroda AR, O'Donnell M. Strain rate imaging using two-dimensional speckle tracking. IEEE Trans Ultrason Ferroelectr Freq Control 2001;48:1111-23
17. Amundsen BH, Helle-Valle T, Edvardsen T, Torp H, Crosby J, Lyseggen E, et al. Noninvasive myocardial strain measurement by speckle tracking echocardiography: validation against sonomicrometry and tagged magnetic resonance imaging. J Am Coll Cardiol 2006;47:789-93.
18. Korinek J, Wang J, Sengupta PP, Miyazaki C et al. Two-dimensional strain—a Doppler-independent ultrasound method for quantitation of regional deformation: validation in vitro and in vivo. J Am Soc Echocardiogr 2005;18:1247-53.
19. Cho GY, Chan J, Leano R, Strudwick M, Marwick TH. Comparison of two-dimensional speckle and tissue velocity based strain and validation with harmonic phase magnetic resonance imaging. Am J Cardiol 2006; 97:1661-6.
20. Bansal M, Cho GY et al. Feasibility and accuracy of different techniques of two-dimensional speckle based strain and validation with harmonic phase magnetic resonance imaging. J Am Soc Echocardiogr 2008;21:1318-25.

21. Tanabe M, Suffoletto MS, Pinsky MR, et al: Validation of novel echocardiographic speckle tracking radial strain to assess ventricular dyssynchrony: Comparison with angle corrected tissue Doppler strain imaging. *J Am Coll Cardiol* 2007;49 (Suppl A):806–808, 100A.
22. Suffoletto MS, Dohi K, Cannesson M, Saba S. Novel speckle-tracking radial strain from routine black-and-white echocardiographic images to quantify dyssynchrony and predict response to cardiac resynchronization therapy. *Circulation*. 2006;113(7):960-8.
23. Delgado V, Ypenburg C, van Bommel RJ, Tops LF, Marsan NA, et al. Assessment of left ventricular dyssynchrony by speckle tracking strain imaging. comparison between longitudinal, circumferential, and radial strain in cardiac resynchronization therapy. J Am Coll Cardiol 2008;51:1944-52.
24. Helm RH, Leclercq C, et al. Cardiac dyssynchrony analysis using circumferential versus longitudinal strain: implications for CRT. Circulation 2005;111:2760-7.
25. Mele D, Toselli T, Capasso F, Stabile G et al. Comparison of myocardial deformation and velocity dyssynchrony for identification of responders to cardiac resynchronization therapy. Eur J Heart Fail 2009;11:391-9.
26. Kass DA. An epidemic of dyssynchrony. But what does it mean? JAm Coll Cardiol 2008;51:12-7.
27. Takemoto Y, Hozumi T et al. Beta-blocker therapy induces ventricular resynchronization in dilated cardiomyopathy with narrow QRS complex. J Am Coll Cardiol 2007;49: 778-83.
28. Miyazaki C, Powell BD, Bruce CJ, et al. Comparison of echocardiographic dyssynchrony assessment by tissue velocity and strain imaging in subjects with or without systolic dysfunction and with or without left bundle-branch block. *Circulation*. 2008;117(20):2617-25.
29. Lim P, Buakhamsri A, Popovic ZB, et al. Longitudinal strain delay index by speckle tracking imaging: a new marker of response to cardiac resynchronization therapy. *Circulation*. 2008;118(11):1130-7.

30. Wiggers CJ. The muscular reactions of the mammalian ventricles to artificial stimuli. Am J Physiol 1925;73:346-78.
31. AbrahamWT, Delurgio DB, Leon AR, Loh E, et al. Cardiac resynchronization in chronic heart failure. N Engl J Med 2002; 346:1845-53.
32. Cazeau S, Leclercq C, Lavergne T et al. Effects of multisite biventricular pacing in patients with heart failure and intraventricular conduction delay. N Engl J Med 2001;344:873-80.
33. Cleland JG, Daubert JC et al. The effect of cardiac resynchronization on morbidity and mortality in heart failure. N Engl J Med 2005;352:1539-49.
34. Achilli A, Sassara M, Ficili S et al. Long-term effectiveness of cardiac resynchronization therapy in patients with refractory heart failure and ''narrow'' QRS. J Am Coll Cardiol 2003;42:2117-24.
35. Molhoek SG, VanDerWall EE, SchalijMJ. QRS duration and shortening to predict clinical response to cardiac resynchronization therapy in patients with end-stage heart failure. Pacing Clin Electrophysiol 2004;27:308-13.
36. Bax JJ, Ansalone G et al. Echocardiographic evaluation of cardiac resynchronization therapy: ready for use ? A critical appraisal. J Am Coll Cardiol 2004;44:1-9.
37. Beshai JF, Grimm RA, Nagueh SF, Baker JH II, Beau SL, Greenberg SM, et al. Cardiac-resynchronization therapy in heart failure with narrow QRS complexes. N Engl J Med 2007;357:2461-71.
38. Teske AJ, De Boeck BW, Melman PG, et al. Echocardiographic quantification of myocardial function using tissue deformation imaging, a guide to image acquisition and analysis using tissue Doppler and speckle tracking. *Cardiovascular ultrasound.* 2007;5:27.
39. Weidemann F, Jamal F: Myocardial function defined by strain rate and strain during alterations in inotropic states and heart rate. *Am J Physiol Heart Circ Physiol* 2002, 283:H792-H799.
40. Greenberg NL, Firstenberg MS: Doppler- Derived Myocardial Systolic Strain Rate Is a Strong Index of Left Ventricular Contractility. *Circulation* 2002, 105:99-105.

41. Abraham TP, Dimaano V. Role of tissue Doppler and strain echocardiography in clinical practice. Circulation 2007;116: 2597-609.
42. Yip G, Abraham T, Belohlavek M, Khandheria BK. Clinical applications of strain rate imaging. J Am Soc Echocardiogr 2003;16:1334-42.
43. Covell JW. Tissue structure and ventricular wall mechanics. Circulation 2008;118:699-701.
44. Sengupta PP, Tajik AJ, Chandrasekaran K, Khandheria BK. Twist mechanics of the left ventricle: principles and application. JACC Cardiovasc Imaging 2008;1:366-76.
45. Isaaz, K, Thompson, A, Ethevenot, G, et al. Doppler echocardiographic measurement of low velocity motion of the left ventricular posterior wall. Am J Cardiol 1989; 64:66.
46. Cheuk-Man Y, Sanderson JE, Marwick TH, Oh JK. Tissue Doppler imaging a new prognosticator for cardiovascular diseases. J Am Coll Cardiol 2007;20(3):234–43.
47. Urheim S, Edvardsen T, Torp H, et al. Myocardial strain by Doppler echocardiography: validation of a new method to quantify regionalmyocardial function. Circulation 2000;102:1158–64.
48. Perk G, Tunick PA, Kronzon I. Non-Doppler two-dimensional strain imaging by echocardiography — from technical considerations to clinical applications. J Am Soc Echocardiogr 2007;49(19):1903–14.
49. Behar V, Adam D, Lysyanski P, et al. Improving motion estimation by accounting for local image distorsion. Ultrasonics 2004;43(1):57–65.
50. Korinek J, Kjaergaard J, Sengupta PP, et al. High spatial resolution speckle tracking improves accuracy of 2-dimensional strain measurements. J Am Soc Echocardiogr 2007;20(2):165–70.
51. Yu CM, Zhang Q, Fung JW. A novel tool to assess systolic asynchrony and identify responders of cardiac resynchronization therapy by tissue synchronization imaging. *J Am Coll Cardiol.* 2005;45:677– 684.

GLOSSARY

2D	-	Two dimensional
CCF	-	Congestive Cardiac Failure
CRT	-	Cardiac Resynchronization Therapy
DTI	-	Doppler Tissue Imaging
EDV	-	End Diastolic volume
EF	-	Ejection Fraction
ESV	-	End Systolic volume
LA	-	Left atrium
LBBB	-	Left bundle branch block
LV	-	Left Ventricle
LVIDd	-	Left Ventricular internal end diastolic diameter.
LVISd	-	Left Ventricular internal end systolic diameter
MD-TDI	-	12 segment maximum delay of time to peak velocity
MR	-	Mitral Regurgitation
NYHA	-	New York Heart Association
PASP	-	Pulmonary artery Systolic Pressures
QRSD	-	QRS Duration
ROC	-	Receptor Operator Curve
SD	-	Standard Deviation
S-L wall delay	-	Septal-lateral delay of time to peak velocity (TSL)
SR	-	Strain Rate
STE	-	Speckle tracking echocardiography
TDI	-	Tissue Doppler Imaging
Yu index	-	SD in 12 segments of time to peak velocity by TDI


Master Chart

Demographic features																ECHO							
Name	Age	sex(1) Male, 2) Female)	case(1- LBBB with LV dysfunction, 2-LBBB normal LV function, 3- IVCD with LV dysfunction, 4-Normal control)	functional class	h/o ACS	ACS(0- no, 1- AWMI, 2- IWMI, 3-USA)	CCF(0- NO, 1- SINGLE EPISODE, 2- MULTIPLE)	INTERVENTIONS [illegible]	PTCA+CABG)	DM	HTN	DLP	SMK	FHOCAD	QRSD	LVIDD	LVISD	LA	MR(0--NONE, 1- MILD, 3- MOD TO SEVERE)	LVEDV	LVESV	EF	PAH (30-44 mild, 45-59 mod, >=60 severe)
Narayanan unni	63	1	1	4	0	0	1	0	0	1	0	1	0	0	140	52	45	44	3	94	68	13	36
Krishna pilai	53	1	1	3	0	0	0	0	0	0	0	1	0	0	130	70	63	34	1	169	130	20	16
Vijaya laxmi	76	2	1	4	1	1	2	1	2	1	1	1	0	1	160	60	44	44	1	164	120	27	20
Jessintha mohan	51	2	1	3	0	0	0	0	0	1	0	1	0	0	140	51	40	36	1	150	91	31	35
Modeen koya	62	1	1	2	0	0	0	0	0	1	0	1	0	0	140	61	49	37	1	197	139	29	20
Mohd elias	70	1	1	2	1	1	0	0	0	1	0	1	1	0	140	57	46	46	1	129	85	33	20
Surendran	56	1	1	3	1	2	1	1	1	1	1	1	1	1	140	50	46	42	1	124	79	35	60
Jessintha	60	1	1	3	0	0	0	0	0	1	1	0	1	0	150	58	49	44	1	145	100	30	35
vellammal	68	2	1	2	0	0	0	0	0	0	1	1	0	1	160	74	61	48	3	190	140	25	32
Kunjumon	49	1	1	3	1	1	0	1	2	0	1	1	1	0	140	60	43	43	1	165	110	35	20
Rahim	47	1	1	4	0	0	1	1	1	1	1	1	0	0	160	59	43	40	3	216	149	32	20
John PV	78	1	1	2	1	1	0	0	0	1	1	1	1	1	180	64	54	41	1	126	88	30	45
Basheer	45	1	2	2	0	0	0	1	2	0	1	1	1	1	140	40	26	36	1	95	40	55	20
subramanian	67	1	2	2	0	0	0	1	3	0	0	1	0	1	160	50	32	39	1	93	45	50	20
Subaida PA	46	2	2	2	0	0	0	0	0	0	1	1	0	0	150	49	23	37	1	83	33	60	22

Vasantha sivarajan	54	2	2	2	1	3	0	0	0	1	1	1	0	0	130	48	28	34	1	80	30	60	30
Shashidharan	55	1	2	2	0	0	0	0	0	0	0	1	1	0	140	62	40	39	3	111	50	55	18
Sasikala M	45	2	2	2	1	3	0	0	0	0	0	1	0	1	140	40	28	35	1	80	30	62	18
Raveendran K	53	1	2	2	0	0	0	0	0	0	1	1	0	0	140	52	36	35	1	79	26	67	18
Jainamma joseph	66	2	2	2	0	0	0	0	0	0	0	1	0	0	140	55	33	37	1	83	35	58	22
Karunakaran nair	79	1	2	2	0	0	0	0	0	0	1	1	1	0	160	53	38	38	1	84	40	52	20
Sarojini amma	70	2	2	2	0	0	0	0	0	1	1	1	0	0	150	52	37	38	1	90	37	54	22
Chandrika	64	2	2	2	0	0	0	0	0	1	0	1	0	0	160	59	28	37	1	62	23	80	20
Mariamma thomas	51	2	3	2	0	0	0	0	0	0	1	1	0	1	90	51	38	36	0	85	67	33	20
Shamshudeen	41	1	3	4	1	1	1	1	1	0	0	1	1	0	100	59	47	45	3	125	93	35	54
Mohd Haneefa	67	1	3	2	0	0	0	0	0	1	0	1	0	0	100	60	53	38	1	147	105	28	19
Rajappan TR	60	1	3	2	0	0	0	0	0	0	0	1	1	0	100	64	32	40	3	150	98	35	26
Retnemma	58	2	3	3	0	0	1	0	0	1	1	1	0	0	110	71	65	42	1	173	136	21	28
Mytheen	36	1	3	2	0	0	0	0	0	0	0	0	1	0	80	64	58	44	1	150	120	17	45
Rajesh	24	1	3	4	0	0	1	0	0	0	0	0	0	0	90	66	61	47	3	179	150	16	35
Subbaiaha	45	1	3	3	0	0	0	0	0	0	1	1	1	0	100	64	50	35	1	158	108	31	32
Kannan	29	1	3	4	0	0	1	0	0	0	0	0	0	0	110	70	63	30	1	145	115	20	25
Amanulla	50	1	3	4	0	0	1	0	0	1	0	1	1	0	100	70	50	47	3	140	85	35	60
Satheesh	40	1	4	0	0	0	0	0	0	0	0	0	0	0	80	44	26	31	1	90	34	66	18
Subair	52	1	4	2	0	0	0	0	0	1	1	1	1	0	90	56	27	35	1	92	34	63	18
Suresh babu	38	1	4	0	0	0	0	0	0	0	0	1	1	0	80	46	28	32	0	80	30	63	20
Rajan	45	1	4	2	0	0	0	0	0	0	1	1	0	1	90	44	27	31	1	74	35	58	18
Rajamma	52	2	4	2	0	0	0	0	0	1	0	1	0	1	80	43	26	30	0	72	36	50	18
Amith	30	1	4	0	0	0	0	0	0	0	0	1	1	0	90	45	25	39	0	98	31	68	18
Amuthan	32	1	4	0	0	0	0	0	0	0	0	1	0	0	100	45	28	26	1	85	32	67	20
Dixon	40	1	4	2	0	0	0	0	0	1	1	1	0	1	100	43	26	30	2	85	33	66	20
Shihab	36	1	4	0	0	0	0	0	0	0	0	1	1	1	90	44	28	27	1	69	23	67	18
Aju dev	24	1	4	0	0	0	0	0	0	0	0	0	0	0	80	41	22	23	0	70	25	70	16

	TDI			Speckle based		
Name	YU INDEX (≥33- abnormal)	S L WALL DELAY (normal <50, significant ≥65)	12 SITE MAX DELAY (normal <90, significant ≥100)	speckle dyssynchrony index	Strain delay index (significant ≥25)	SEGEMNTS NOT ANALYSED
Narayanan unni	34.8	102	120	72.7	34.1	0
Krishna pilai	35.85	56	99	69.96	21.16	1
Vijaya laxmi	55.7	61	154	65.15	29.53	4
Jessintha mohan	45.2	98	125	69.5	22.333	0
Modeen koya	50.7	92	170	63.8	35.44	0
Mohd elias	63	122	191	63.31	23	0
Surendran	58	163	161	84.26	25.7	0
Jessintha	40.88	56	119	76.8	22	1
vellammal	34.13	44	103	88.56	32.38	2
Kunjumon	53.025	107	165	87.5	27.22	1
Rahim	37.94	77	110	61.62	26	0
John PV	57.9	106	163	120.26	29.5	1
Basheer	30.51	73	101	52.74	17.88	1
subramanian	49.1	75	150	62.3	10	0
Subaida PA	41.65	69	136	50.3	23	0

Vasantha sivarajan	39.72	74	94	54.68	18.8	0
Shashidharan	39.41	64	108	32.72	25.71	1
Sasikala M	36.38	58	108	44.8	12.6	1
Raveendran K	33.92	70	94	55.63	22.17	1
Jainamma joseph	37.15	75	97	59.84	26.62	0
Karunakaran nair	38.42	101	107	67.56	11.5	0
Sarojini amma	43.6	77	140	43.26	23	4
Chandrika	43.76	101	105	53.76	22	0
Mariamma thomas	41.63	68	110	67.7	12	2
Shamshudeen	42.24	31	141	41.07	18.95	2
Mohd Haneefa	23.21	37	82	57.39	16.32	4
Rajappan TR	25.85	59	69	51.78	24.6	0
Retnemma	48.9	10	144	50.73	26.14	0
Mytheen	18.27	58	59	46.2	14.7	1
Rajesh	26.96	70	87	38.04	16.36	0
Subbaiaha	42.7	28	135	44.4	24.2	1
Kannan	40.4	52	129	35.13	24.1	2
Amanulla	27.78	69	74	54.3	18.3	0
Satheesh	21.54	60	64	27.04	8.7	0
Subair	32.76	27	75	38.81	7	0
Suresh babu	28.63	16	85	33.5	8	0
Rajan	11	17	34	48	8.23	2
Rajamma	23.78	55	75	33.21	8.95	1
Amith	19.9	47	51	41.37	7.2	0
Amuthan	16.85	36	50	34.79	11.06	0
Dixon	14.3	29	41	39.64	6	0
Shihab	26.67	40	66	29.64	9.5	0
Aju dev	30.33	73	76	36.95	8.2	1