"Intraoperative Assessment Of Transient And Persistent Regional Left Ventricular Wall Motion Abnormalities In Patients Undergoing Coronary Revascularization Surgery Using Real-time 3D Echocardiography"

A Prospective Observational Study

PROJECT REPORT

Submitted during the course of
DM Cardiothoracic and Vascular Anaesthesia

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Jan 2014 – Dec 2016

DECLARATION

*I, **Dr. Neelam Aggarwal,** hereby declare that the project in this book was undertaken by me under the supervision of the faculty, Department of Anaesthesiology (Cardiothoracic & Vascular Anaesthesia), Sree Chitra Tirunal Institute for Medical Sciences and Technology.*

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ACKNOWLEDGEMENT

*I am deeply indebted to my guide **Prof. Koniparambil P Unnikrishnan** for his tremendous encouragement and constructive criticism throughout the course of this study.*

*I am very grateful to my co-guide **Prof. P.R. Suneel** for his constant encouragement and valuable suggestions at all stages of this study.*

*I owe my sincere thanks to **Prof. Rupa Sreedhar** who showered her unconditional help and faith in me.*

*I express my gratitude to **Prof. Jayakumar K** for his guidance and motivation throughout the course of my study.*

*I am always indebted to my **Parents** for their blessings and support in completing the study.*

*I owe heartfelt thanks to **Monika Aggarwal** (my sister) and **Pankaj Aggarwal** (my brother) who have helped me in a multitude of ways for making me feel confident during the study.*

*I am also grateful to **Prof. Thomas Koshy and Shrinivas .V. Gadhinglajkar** for their cooperation and motivation during every phase of my study.*

*I owe a special thanks to my friend **Dr Indranil Biswas** for his unconditional help during this study.*

I sincerely thank all my colleagues of the department and anaesthesia technicians for their constant help.

I am deeply indebted to my patients for their cooperation and sacrifices for the greater good of fellow human beings.

*Dedicated to my beloved sister **Monika Aggarwal** and my family*

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List of Abbreviations

S.NO.	SYMBOL	EXPANSION
1.	CAD	Coronary artery disease
2.	RWMA	Regional wall motion abnormality
3.	CABG	Coronary artery bypass grafting
4.	LV	Left ventricle
5.	2D	2-dimensional
6.	TEE	Transesophageal echocardiography
7.	RT3DE	Real time 3 dimensional echocardiography
8.	SDI	Systolic dyssynchrony index
9.	CRT	Cardiac resynchronization therapy
10.	PMI	Parametric motion imaging
11.	WMSI	Wall motion score index
12.	MI	Myocardial infarction
13.	CPB	Cardio-pulmonary bypass
14.	SWMAs	Segmental wall motion abnormalities
15.	ME	Mid esophageal
16.	4 chamber	4C
17.	2 chamber	2C
18.	LAX	Long axis
19.	TG	Trans-gastric
20.	SAX	Short axis
21.	AHA	American Heart Association
22.	ASE	American Society of Echocardiography
23.	SWT	Segmental wall thickening
24.	TDI	Tissue Doppler imaging
25.	STI	Speckle-tracking imaging
26.	FV	Full volume
27.	CMR	Cardiac magnetic resonance
28.	TSI	Tissue synchronization imaging
29.	MPR	Multiplanar reconstruction

30. TTE Transthoracic echocardiography

S.NO.	SYMBOL	EXPANSION
31.	V-t curve	Volume-time curve
32.	MSV	Minimal systolic volume
33.	LVEF	Left ventricular ejection fraction
34.	EDV	End diastolic volume
35.	ESV	End systolic volume
36.	Tmsv 16-diff	Maximal difference in time to reach MSV among 16 segments
37.	Tmsv 16–SD	Standard deviation of time to reach MSV for 16 segments
38.	PMI	Parametric motion imaging
39.	Exc avg	Average value of excursion deviation
40.	EAS	Early activating segments
41.	MR	Mitral regurgitation
42.	LAD	Left anterior descending
43.	ADR	Adrenaline
44.	LBBB	Left bundle branch block
45.	RMS	Root mean square

INTRODUCTION

Introduction

Acute or chronic underperfusion of myocardium in patients with coronary artery disease (CAD) results in depressed contractile state of myocytes which is expressed as regional wall motion abnormality (RWMA).[1] Wall motion abnormality is traditionally classified as hypokinesia, akinesia and dyskinesia. Hypokinetic segments with preserved wall thickness and echogenicity represent reversible myocardial injury, whereas on the other hand, akinetic or dyskinetic segments with wall thinning and increased echogenicity generally indicate irreversible myocardial injury. However, the latter may occur due to reversible non-lethal injury. Irreversible injury happens due to infarction or scarring, while reversible injury is due to ischemia. Myocardial contractile dysfunction can also be described as having transient or persistent RWMA depending upon duration and severity of ischemic insult.[2] Prolonged duration of ischemia leads to persistent RWMA which can be either the hibernating myocardium retaining the potential of complete functional recovery after revascularization or the necrosed myocardium with no scope of recovery even after re-establishment of coronary flow. Due to short term ischemia (less than 20 minutes) followed by reperfusion, a transient state of contractile dysfunction, known as stunning may develop, and it persists for hours or days. It returns to normal function either spontaneously or with inotropic support.[3-5] Therefore, both stunning and hibernation contribute to dysfunctional, yet viable myocardium.

Coronary artery bypass grafting (CABG) is the mainstay for management of the patients suffering from CAD. Myocardial function recovery after revascularization depends upon presence of viable myocardium. Many clinicians usually subject patients to viability testing in the pre-operative period and perform CABG for only those patients who have at least 25-30% of viable myocardium.[4,5] Quantification of

regional function is of considerable importance not only in the pre-operative period for planning and execution of surgery but also when the surgery is going on, so that timely medical or surgical intervention is guaranteed. Monitoring of global and regional function of left ventricle (LV) in patients undergoing CABG by 2-dimensional (2D) transesophageal echocardiography (TEE) is an estabilished practice but the role of intraoperative real-time 3 dimensional echocardiography (RT3DE) to assess regional myocardial function is not yet fully explored.

RT3DE can be employed to identify intra-ventricular mechanical dyssynchrony as one of the parameter to assess regional myocardial dysfunction. Assessment of systolic dyssynchrony index (SDI) by RT3DE has been recently adopted to predict the response of cardiac resynchronization therapy (CRT) in patients having chronic heart failure due to dilated cardiomyopathy.[6,7] In context of ischemic cardiomyopathy, Penicka M et al have emphasized the need to assess LV dyssynchrony in addition to myocardial viability in patients undergoing CABG.[8] They studied the patients having moderate systolic heart failure with narrow QRS duration due to ischemia and discovered the presence of LV mechanical dyssynchrony in this patient population, which could affect their outcome of revascularization. It has been proven that the mechanical delay of LV segmental contractions lead to intra-ventricular dyssynchrony in the absence of conduction disturbance of electrical impulses.[9]

Therefore, systolic dyssynchrony index may reflect tissue contractile state and diagnose the presence of regional contractile dysfunction.

There has been further technological advancement in 3D technology such as Parametric Motion Imaging (PMI) in the recent past to quantitate endocardial wall motion more precisely. However, these techniques are still underutilized in our routine practice.

The present study was done with the aim to investigate:

1) The **feasibility** of RT3DE to identify and quantify regional contractile dysfunction in patients undergoing coronary revascularization.

2) The **comparability** of RWMA assessment by RT3DE derived SDI and 2D TEE derived wall motion score index (WMSI).

AIMS AND OBJECTIVES

Aims and Objectives

Primary objectives:

1. To identify LV mechanical dyssynchrony using RT3DE in patients having ischemic cardiomyopathy undergoing CABG.
2. To quantitatively assess transient and persistent regional wall myocardial abnormalities (RWMAs) using RT3D echocardiographic parameters.
3. To assess the effect of myocardial revascularization and inotropic support on the resolution of LV dyssynchrony and RWMAs.

Secondary objective:

1. To study the correlation of regional wall motion assessment by two imaging modalities: 2D WMSI and 3D SDI.

REVIEW OF LITERATURE

Review of Literature:

In patients with coronary artery disease, RWMAs may result in the form of decreased contraction (hypokinesia), absent movement (akinesis) or paradoxical movement (dyskinesis) from regional myocardial ischemia or from myocardial infarction (MI) and scar formation.[1] These RWMAs are thought to be characteristic of ischemic cardiomyopathy and indicate severity of regional myocardial dysfunction. Depending upon duration and severity of ischemia, these RWMAs can return back to normal tissue state of contraction either spontaneously or following appropriate interventions. In case of irreversible injury due to necrosis, RWMAs will persist even if flow to the myocardium is re-established. Over a period of three decades, there has been aggressive research on pathogenesis, identification and subsequent management of regional LV dysfunction, which has drastically improved the prognosis of patients with CAD. In this context, it was realized that description of regional wall motion based on the visual assessment of state of contraction (hypokinesia, akinesia or dyskinesia) was not sufficient and therefore, attempts were made to identify the reversibility of myocardial dysfunction to determine the necessity of revascularization and predict the functional recovery. The term viability came into light, which described the dysfunctional myocardium subtended by diseased coronary arteries with limited or absent scarring retaining the potential for functional recovery.[4,5] Later on, two tissue states were discovered to identify dysfunctional but viable myocardium, and were termed as stunning and hibernation. Braunwald and Kloner[10] defined stunned myocardium as a state where despite of early and complete return of blood flow, there is transient contractile dysfunction that eventually recovers. Stunning occurs in the following situations - unstable angina, exercise-induced ischemia,

reperfused acute MI and after cardiac surgery due to aortic cross clamping and cardioplegia.[3] In the former three situations, regional LV dysfunction, and in the last case global contractile dysfunction are common consequences. Now with the advancement in techniques of myocardial protection, stunning after separation from cardio-pulmonary bypass (CPB) may be less severe. In patients with CAD, repeated episodes of demand ischemia may lead to cumulative stunning. There are several hypotheses to explain this post reperfusion contractile dysfunction observed in stunned myocardium including deleterious effects of oxyradicals on the contractile elements and abnormal myofilament sensitivity to calcium. Hibernation was originally defined by Rahimtoola as an adapted state of myocardium due to chronic underperfusion leading to depressed contractile function in order to match the decreased supply of substrates and oxygen.[11] Some authors have also proposed that hibernation usually develops following recurrent episodes of severe ischemic insult and is in essence, a result of repetitive periods of stunning.

Therefore, a patient with CAD is a clinical entity, where both stunning and hibernation often co-exist and lead to transient and persisting regional wall motion abnormalities, represented by hypokinesia, akinesia or dyskinesia. It is noteworthy that this is in contrast to the usual belief that akinetic or dyskinetic segments will always represent non-viable or infarcted myocardial tissue. The rationale to differentiate all these abnormal tissue states is that they respond in a different manner to therapeutic interventions and carry different prognosis. In stunning, functional recovery is a rule after hours or days depending upon duration and severity of ischemia. Since by definition, stunning has normal blood flow, it does not require any reperfusion therapy and contractile dysfunction usually recovers spontaneously. However, if it is severe, impairing global LV function, it can be reversed with

inotropic agents.[3] The therapy of hibernating myocardium is to restore blood flow to the hypoperfused tissue by revascularization strategies and the time to recovery of function may be immediate for majority of patients but others may follow a much more prolonged course of recovery. Infarcted myocardium is irreversibly damaged tissue and does not respond to any intervention.

With the increasing understanding of heterogeneous response to revascularization, many clinicians are subjecting patients to viability testing in the pre-operative period either by stress echocardiography or nuclear imaging modalities.[4,5] In dobutamine stress echocardiography (DSE), by administering escalating doses of dobutamine, tissues with inotropic contractile reserve are recognized as hibernating and stunned myocardium. Hibernating tissue may exhibit a biphasic response, contractile performance improving at lower doses (5–10 μg/kg/min) but declining at higher doses (>15μg//kg/min). It happens when the metabolic demand overwhelms the tissue's capacity to respond causing perfusion-contraction mismatch. In hibernating myocardium, the increase in contractile function is at the expense of metabolic recovery whereas in stunned myocardium no metabolic deterioration occurs during inotropic stimulation.

In view of advanced hemodynamic monitoring of global as well as regional myocardial function of the patients undergoing CABG, anesthesiologists have lately realized the value of intraoperative TEE. They have been aiming to assess pre-CPB RWMAs and post revascularization improvement in previously jeopardized myocardium or appearance of any new RWMA for evaluation of the revascularization procedure. The presence of new segmental wall motion abnormalities (SWMAs) at the end of surgery may guide the surgeon to revaluate the patency of the coronary bypass graft. In addition, these may predict a complicated postoperative course.

Previous studies have documented various 2D and 3D TEE measures to assess intraoperative regional contractile dysfunction in patients undergoing CABG. The most commonly employed method is LV segmental wall motion analysis, which is obtained from six TEE views: Mid esophageal (ME) 4 chamber (4C), ME 2 chamber (2D), ME LV long axis (LAX), trans-gastric (TG) basal short axis (SAX), TG mid papillary and TG apical views. Segmental LV wall motion is analyzed using a 17-segment American Heart Association (AHA) model and computing WMSI as described by American Society of Echocardiography (ASE) guidelines. It is a semi-quantitative measure that is highly dependent on image quality. It is recommended that at least 50% of the endocardial and epicardial borders should be visible in a segment to be graded as normal segment. In addition, to grade a segment as abnormal, approximately 33% of the endocardial and epicardial borders have to be visible. However, if the epicardium is not visible, a segment still can be considered adequate for analysis if 90% of the endocardial border is completely visible throughout the cardiac cycle. Segments not fulfilling these criteria are graded as 0 or no view.[12] WMSI is obtained by summation of the score of each segment divided by the number of myocardial segments examined. Higher the values of WMSI, more is the presence of hibernating or necrotic segments. There are many limitations of this method. Firstly, it is a subjective parameter and subtle differences in wall motion are often missed visually. During the surgery, evidence of ischemia is defined as the worsening of segmental wall motion by two or more grades in two or more segments. Therefore, marked changes in wall motion are required to maintain a high specificity for diagnosing intraoperative ischemia.[12] Secondly, it does not differentiate viable from the non-viable tissue. Thirdly, it may be influenced by an unmasked scar caused by changes in afterload and the tethering effect caused by a neighboring infarcted

segment. Lastly, it may appear that there is abnormal paradoxical motion of the inter-ventricular septum during the surgery inducing spurious high values of wall motion index. This paradoxical septal motion is due to the exaggerated systolic anteromedial translation of the entire heart within the chest.[13] Assigning wall motion score with an internal frame of reference or floating-axis analysis system may rectify the error.

Other two frequently used semi-quantitative measures of detecting regional LV abnormalities are: segmental wall thickening (SWT) and endocardial wall shortening. Previous studies suggested that analysis of regional thickening is much more precise than segmental wall motion for determination of viability.[13,14] Furthermore, measurement of systolic thickening is relatively unaffected by translation motion of heart during the cardiac and respiratory cycle. SWT is analyzed in TG mid papillary view. The wall thickness is measured by tracing endocardial (excluding papillary muscles) and epicardial borders within each segment (in the middle of the segment and along the radius drawn through the midsection) during end-diastole and end-systole. The anterior and posterior papillary muscles provide a fixed external frame of reference for determination of the midsection of the left ventricular cavity. The percentage SWT is calculated by subtracting diastolic SWT from systolic SWT and dividing the difference by systolic SWT.[14] The ASE guidelines provide a scoring system: score 1 = >30% increase in wall thickness during systole, score 2 = 10 - 30%, score 3 = <10%, score 4 = no wall thickening and a score of 5 for wall thinning during systole. The prerequisite for all the above-mentioned methods is the acquisition of high quality images with sharp definition of endocardial and epicardial borders and inability to do so, lead to errors in analysis.

With the known limitations of these semi-quantitative methods, there have been continued efforts to further refine the assessment of SWMA. The trend has shifted to

the adoption of modalities that are more precise and objective. Recently, tissue Doppler imaging (TDI) has been utilized to measure segmental myocardial velocity and displacement to assess strain and strain rate.[15] However, the method inherits the drawbacks of substantial angle dependency. Moreover, TDI strain values may get altered due to tethering by infarcted segments. The role speckle-tracking imaging (STI) has also been explored, which uses acoustic myocardial markers and semiautomated border detection to measure strain and strain rate.[15] STI is more promising than TDI in terms of reliability and precision but it is time consuming and requires expertise.

Currently 3D echocardiography is in vogue and there are recent advances in computer processing and transducer construction techniques (matrix array probe) that provide real time images.[16] Various software programs (Q-lab /Tom Tec) are now readily available to analyze 3D datasets of the LV and to generate highly accurate analysis of LV morphology and function without any geometric assumptions (comparable to gold standard of cardiac magnetic resonance). An ECG gated (4–7 beats) full volume (FV) loop has to be acquired first to obtain a large 3D dataset. It is created from many subvolumes of LV that are stitched together during briefly held respiration and synchronized to one cardiac cycle with overlapping ECG traces. FV acquisition begins in a biplane preview and frame rate is kept above 20 Hz to achieve good temporal resolution. Optimization of a 3D FV image necessitates gain (50%) and compression (50%) adjustment in 2D image before FV acquisition. In this way, a pyramidal 3D volume dataset of 90 °X 90 ° at a frame rate of 20–25 Hz is obtained to allow comprehensive analysis of the LV.[16] One study published by Corsi et al suggested assessment of RWMAs by RT3DE and validated the results against cardiac magnetic resonance (CMR).[17] The authors used a customized software designed to

semi-automatically detect and segment the endocardial surface. After identification of long axis of LV, the radial distance between the long axis and each surface voxel was computed and averaged for each segment. Subsequently, regional wall motion (RWM) was calculated as the difference between end diastolic and end systolic radial dimensions. They concluded that the agreement between RT3DE and CMR values was directly related to RT3DE image quality and contrast enhancement improved the accuracy of RWM quantification. The prerequisite for obtaining accurate result by RT3DE remained the acquisition of high quality 2 dimensional images.

Assessment of intra-ventricular dyssynchrony is increasingly recognized as an important aspect of cardiac assessment in different clinical scenarios. Dyssynchrony may occur during ventricular contraction (systolic dyssynchrony) or during relaxation (diastolic dyssynchrony).[18] Systolic dyssynchrony is defined as uncoordinated timing of contraction in different regions (or segments) of the myocardium that can be due to delay in conduction of electrical impulses or delay in mechanical contraction. There are several imaging methods to evaluate cardiac dyssynchrony such cardiac MRI, electroanatomic mapping by measuring the changes of regional volumetric curves and echocardiography.[18] Echocardiography has certain advantages over other techniques being easily accessible, non invasive and no risk of radiation exposure. A number of echocardiographic modalities have been developed to assess intraventricular dyssynchrony such as TDI, strain rate imaging, tissue synchronization imaging (TSI), displacement imaging and RT3DE.[18,19] In order to measure SDI by RT3DE, the first step is to export the 3D datasets to a customized in-built software to calculate volumes of the ventricle at multiple points during the cardiac cycle.[16,19] A semi-automated endocardial border detection process is followed after proper alignment of multiplanar reconstruction (MPR) planes and assignment of five reference points at the mitral

annulus and apex. Finally with the sequential analysis of data, the software creates a mathematical model or ‘‘cast’’ of LV, which is subdivided into 17-AHA segments according to transthoracic echocardiography (TTE) orientation. A series of volume-time (V-t) curves is plotted for each segment over the entire cardiac cycle. These segmental V-t curves provide analysis of regional function in the time domain and determine intra-ventricular dyssynchrony. The basic principle is that in a ventricle with synchronous contraction of all segments, each segment should achieve its minimal systolic volume (MSV) at almost the same point in the cardiac cycle, whereas in a dyssynchronous ventricle there is dispersion in the segmental timings to attain MSV. The degree of dispersion is quantified by systolic dyssynchrony index (SDI), which is measured as the standard deviation of the time in 16 segments to achieve minimum volume normalized to R-R interval.[16,19] The standard values for real-time 3D TTE dyssynchrony parameters in a normal population have been reported.[20] There are few studies in literature that have shown an average correlation between TDI and RT3DE for assessment of dyssynchrony due to ischemia induced myocardial dysfunction.[9,21,22]

The clinical implications of assessment of systolic dyssynchrony have been extensively investigated in ischemic as well as non-ischemic cardiomyopathy. Recent literature review showed that LV dyssynchrony was usually associated in patients with chronic heart failure & wide QRS duration in the context of CRT. Biventricular pacing improved symptoms, ejection fraction and survival by synchronizing the segmental contractions.[23] RT3DE derived systolic dysynchrony index is currently a widely accepted method to identify the responders of CRT in the patients having either ischemic or non-ischemic cardiomyopathy.[6,7] Tanaka H et al demonstrated the association of LV dyssynchrony in significant number of patients of acute heart

failure due to acute onset non-ischemic cardiomyopathy, despite having a narrow QRS duration.[24] They also observed improvement in LV function with resolution of dyssynchrony.

Maruyama Y et al [25] tested RT3DE in order to predict chronic LV remodeling based on dysynchrony measurement after reperfusion therapy in patients who suffered acute MI. They found that increased dyssynchrony immediately after reperfusion correlated negatively with chronic remodeling and positively with LV ejection fraction (LVEF) and the reason being, dyssynchrony reflected stunned and hibernating myocardium, which eventually recovered. In addition, they documented TDI was superior to RT3DE derived dyssynchrony index in identifying transmural infarcted area. It has been studied that in the patients with systolic heart failure due to ischemic heart disease, the prevalence of LV dyssynchrony ranged from 20.8% to 79.6% [8]. Penicka M et al mentioned that revascularization alone was insufficient to resynchronize the LV contraction pattern and therefore, post CABG dyssynchrony was an independent predictor of long-term survival. Similarly, a study by Delgado V et al utilized RT3DE and demonstrated that among patients with acute MI, the most delayed segments were more frequently located at the apical and mid segments corresponding with the SWMAs in the same regions of LV.[9] Therefore, the association of intra-ventricular dyssynchrony and regional contractile dysfunction has been recognized and it is now realized that dyssynchrony can also be measured apart from segmental wall motion, myocardial velocity and strain rate to quantify RWMA. The technical ease, accuracy and reproducibility of the RT3DE have been established.[9] Despite of the presence of enough encouraging evidences, still not much research has been conducted on this subject especially in patients with ischemic heart disease undergoing CABG surgery.

One more interesting application of RT3DE is Parametric Motion Imaging (PMI). It provide us with two color-coded polar maps (bull's eye) that delineate the endocardial wall motion very precisely.[19] The dysfunctional myocardium is represented by red color on both the polar maps with negative values of segmental excursion. Unfortunately, no study has been conducted so far to validate the intraoperative role of RT3D-PMI to identify and quantitate segmental contractile dysfunction in patients with ischemic cardiomyopathy. Therefore, in the current study we aim to utilize RT3DE derived SDI and bull's eye imaging for objective quantification of transient and persisting RWMAs in patients undergoing CABG.

MATERIALS AND METHODS

Methodology

Patient selection

A series of 42 patients scheduled for elective CABG were enrolled prospectively. The Institutional Ethics Committee approved our protocol and written informed consent was obtained from all the patients.

All of them fulfilled the ***inclusion criteria***:

1) Normal sinus rhythm
2) Normal or mild LV dysfunction (LVEF ≥ 40%)
3) With or without RWMA detected on 2D transthoracic echocardiography

***Exclusion Criteria*:**

1. Conduction defects, arrhythmias and permanent pacemaker in situ
2. Moderate to severe LV dysfunction (LVEF <40%)
3. Dilated LV: LV end diastolic dimension > 65mm
   LV end systolic dimension > 45mm
4. Concomitant LV remodeling or valve surgery

Anesthetic Management

After application of standard American Society of Anesthesiologists monitors and invasive arterial blood pressure catheter, all the patients were induced using standard anesthesia protocol of intravenous fentanyl (4-5µg/kg), midazolam (0.1mg/kg) and propofol (1-2mg/kg). Tracheal intubation was facilitated with pancuronium (0.15

mg/kg). Anesthesia was maintained with sevoflurane, fentanyl and midazolam. A comprehensive TEE examination was performed to evaluate LV function using iE33 machine equipped with 3D X7-2t transducer probe (iE33 xMATRIX; Philips Healthcare, Andover, MA). 2D and 3D echocardiographic datasets were acquired at three time intervals:

1) Before going on CPB
2) Immediately after weaning from CPB (before initiating adrenaline infusion)
3) After adrenaline infusion of 0.05 µg/kg/min.

A bolus injection of ephedrine (3-6 mg) was given during separation from CPB whenever systolic blood pressure was less than 90 mmHg or heart rate was less than 60 beats per minute. A mean arterial pressure of 60-70 mmHg and central venous pressure of 8-10 mmHg was maintained to keep constant loading conditions at all the time periods. Patients were excluded if they required temporary pacing and more than one inotropic support, which precluded acquisition of adequate 3D data.

2D Echocardiography (2DE) – Data acquisition

WMSI was recorded in 3 ME views [4 C, 2 C, LV LAX view], and 3 TG views [basal SAX, mid papillary (MP) and apical view] using a 17-segment AHA model. For each segment, endocardial wall motion was scored according to ASE guidelines:

Score 1= Normokinesis

Score 2= Mild hypokinesis

Score 3= Severe hypokinesis

Score 4= Akinesis

Score 5= Dyskinesis

At least 50% of the endocardial and epicardial borders were required to be visible in a segment for adequate analysis of the wall motion.[12] WMSI was obtained by mean of all the scores of 17 segments. Higher the value of WMSI indicated presence of more number of hibernating or necrotic segments. In order to reduce the subjective bias of WMSI, one more independent observer unaware of preoperative TTE findings reviewed the score and the final assigned wall motion score was based on average of the two values.

Real- Time 3D Echocardiography – Data acquisition

A ME 4 chamber full volume (FV) loop was acquired over 4 beats in a briefly held respiration phase at a frame rate between 20 - 25Hz. Gain, compression, time gain compensation and depth settings were optimized to enhance endocardial border detection. Care was taken to include the entire LV cavity within the pyramidal volume scan. Any probe movement, ECG artifacts (stitch artifacts), respiratory movement and electrocautery interference were avoided during image acquisition. We acquired 3 FV loops of LV at each time point and the one with the best image quality was used for off-line analysis. The 3D image was considered unsuitable for analysis if >2 segments could not be visualized or if it contained visible translation artifacts. Thereafter, LV function was quantified by exporting FV 3D datasets to Q-lab version (8.0) software [3DQ Advanced software; Philips Healthcare]. This customized software allowed off-line analysis of global and regional LV function in a simple 3-step process:

1) Alignment of MPR planes passing through LV apex and mid cavity so that they are perpendicular to each other in ME 4 chamber and 2-chamber view

2) Selection of end diastolic and end systolic frame with respect to mitral valve

closure and opening corresponding with ECG

3) Assignment of five reference points, four near the mitral annulus (in 4 Chamber view- septal and lateral walls and in 2 Chamber view- anterior and inferior walls) and fifth one at apex in either of the view.

The computerized algorithm subsequently used these points for semi-automated detection of the endocardial border throughout the cardiac cycle. We manually edited the endocardial borders only in case of gross disparity. A cast of the LV cavity was then created following a mathematical model which was subdivided in 17 wedge shaped segments as described by AHA in TTE orientation, providing volume-time (V-t) curves for each segment over the entire cardiac cycle. The entire process took approximately 60-90 seconds.

The first global V-t curve estimated the global myocardial function (Annexure 1A). It displayed the change in full pyramidal volume of LV as an absolute value over a period of time and computed end diastolic volume (EDV), end systolic volume (ESV) and EF.

In the second regional V-t curve, segmental volume change was plotted against time demonstrating maximum and minimum volumes of that specific segment during the cardiac cycle. The red triangle on the curve indicated the time to reach minimal systolic volume (Tmsv) or time to reach peak systolic excursion for each wedge (Annexure 1B). **In a ventricle with synchronous contraction of all segments, all the red triangles were found clustered together, whereas in a dyssynchronous ventricle there was dispersion of red triangles.**

The maximal difference in the time to reach MSV among the 16 LV segments ***(Tmsv 16- diff)*** was the difference between the timings of the earliest and the most delayed segment. ***It was considered normal if less than 60 msec.*** Apex was excluded from

analysis as it contributed little to ejection. The standard deviation (SD) of time taken to reach MSV for all the 16 segments (***Tmsv 16 -SD***) ***should be less than 32 msec***[16]. In addition, hypo or non-contractile segments could easily be identified based on the pattern of the curve, *while diseased segments had a flattened curve*. This approach has been validated using MRI as the standard of reference [19]. The third normalized V-t curve was the curve between volumes of each segment that contributed to LV pyramidal volume as a percentage (%) of end diastolic volume over a period of time (Annexure 1C). In this curve, red triangle indicated the time to reach minimal systolic volume as a percentage of R-R interval (% R-R) i.e one cardiac cycle. **The degree of dispersion was calculated by measuring the standard deviation of the time to achieve MSV for 16 segments normalized with R-R interval.** This allowed derivation of a systolic dyssynchrony index (SDI), and for patients with no intra-ventricular dyssynchrony, ***SDI should be 3.5 % ± 1.8%.*** [16]

Parametric Motion Imaging (PMI) provided a more fine approach to describe segmental myocardial contraction. More than 800 endocardial data points were used to develop a polar map of the endocardial surface of the LV. Based on an end-diastolic and end-systolic map, two "bull's eye" plots of 17 segments were created representing segmental excursion and timing to reach MSV/ peak systolic exursion for 16 segments (Annexure 1D) in TTE orientation. On excursion plot, endocardial motion was displayed as shades of blue for positive excursion values representing inward motion, red for negative excursion values representing outward motion, and black representing no motion. Blue color on excursion plot was considered as normal regional wall motion. The second polar map (Timing plot) depicted the average time to achieve MSV of each endocardial data point in green color. Events occurring before average motion were displayed in shades of blue, and events occurring after

average motion acquired shades of red to yellow.

Therefore, RT3D assessment of regional wall motion was based on:

1) The quantification of change in LV chamber volume over time from each segment excursion
2) The color change on parametric bull's eye images

The values of 3D EF, Tmsv 16 -diff, Tmsv 16 -SD, SDI and average excursion (Exc avg) were observed at three time intervals.

STATISTICAL ANALYSIS

Statistical Analysis

The continuous variables were expressed as mean ± SD, whereas qualitative variables were expressed as numbers and percentages. Patients were divided into two groups: **Group A** – with no RWMAs and **Group B** - with RWMAs, which were further subdivided into subgroups based on intraoperative TEE findings (figure 2). Using Q – Q plot, the normality for each parameter in every subgroup was checked visually. In the two groups, the parameters with normal distribution were compared by one-way repeated measures of analysis of variance test (ANOVA) at all time points, and the 2-tailed Student t test for paired data between the two time points. The parameters that were not normally distributed and the subgroups with very small sample size were compared using Friedman's test, followed by Wilcoxon signed rank test between individual time points. Definition of LV mechanical dyssynchrony by RT3DE was set with an arbitrary cut-off value of 5.6%, which is in line with the previous studies.[26] Furthermore, the agreement between the presence or absence of RWMA on 2D and RT3DE was expressed as percentage. The concordance between WMSI and SDI was evaluated on scatter plot and Pearson correlation coefficient (r) was calculated for time intervals. P value of less than 0.05 was considered statistically significant. All the data were analyzed using the SPSS version 16.0 statistical package (SPDD, Inc, Chicago, Illinois).

OBSERVATION AND RESULTS

Results

Out of 42 enrolled patients, 4 patients were excluded due to suboptimal image quality and rhythm abnormalities yielding a high feasibility rate of data acquisition by RT3DE in approximately 90% of the cases. The baseline demographic characteristics of the included 38 patients were: 87% men [n=33] and 13% women [n=5]. The mean age of all the patients was 58.16 ± 6.9 years, as shown in table 1.

36% [n=14], 42% [n=16], 84% [n=32] of patients complained of recent acute coronary event, diabetes and hypertension respectively.

VARIABLE (N=38)	MEAN	SD	FREQUENCY	PERCENTAGE (%)
AGE	58.16	6.90		
SEX (MALE) **(FEMALE)**			33 5	86.84 13.16
WEIGHT	66.23	7.09		
HEIGHT	163.76	6.67		

Table 1: Demographic details of the patients. Age, Weight and Height are expressed as mean ± SD, and sex is expressed in number (%).

Pre CPB identification of RWMA

Table 2 enumerates pre-CPB TEE observations of the patients. On the basis of 2DE inspection, patients were divided into two groups:

Group A [n=22] had patients with no RWMAs **(WMSI=1)**

Group B [n=16] consisted of patients noted with RWMAs **(WMSI>1)**

On RT3DE, only 11 patients demonstrated SDI>5.6% whose WMSI values were also abnormal. This resulted in discordance between 2D and 3DE analysis in 5 patients. Further analysis with parametric bull's eye imaging in patients having SDI >5.6%

delineated red and black segments on bull's eye excursion plot representing myocardium with persistent RWMAs. PMI revealed discordance between 2D and 3DE assessment in 2 more patients, as their dysfunctional segments differed on 2D and 3D imaging.

The agreement between the two techniques for identifying RWMA was 82% before establishing CPB. Figure 1 depicts the scatter plot between the pre – CPB values of WMSI and SDI in 38 patients showing an average correlation (r=0.40; p=0.012).

VARIABLE (N =38)	FREQUENCY	PERCENTAGE (%)
3DEF< 50%	9	23.68
WMSI>1	16	42.10
WMSI=1	22	57.90
SDI>5.6%	11	28.95
SDI<5.6%	27	71.05
2D = 3D	31	81.58
2D≠ 3D	7	18.42
WMSI >1 SDI< 5.6%	5	13.16
Segmental analysis Mismatch on bull's eye	2	5.26

Table 2: ***Pre CPB TEE analysis of identifying RWMA by WMSI and SDI.***
Abbreviations:-CPB= cardiopulmonary bypass; TEE= transesophageal echocardiography; RWMA= regional wall motion abnormality; WMSI= wall motion score index; SDI= systolic dyssynchrony index; 3DEF= 3 dimensional ejection fraction; 2D=3D= concordance between WMSI and SDI; 2D≠3D= discordance between WMSI and SDI.

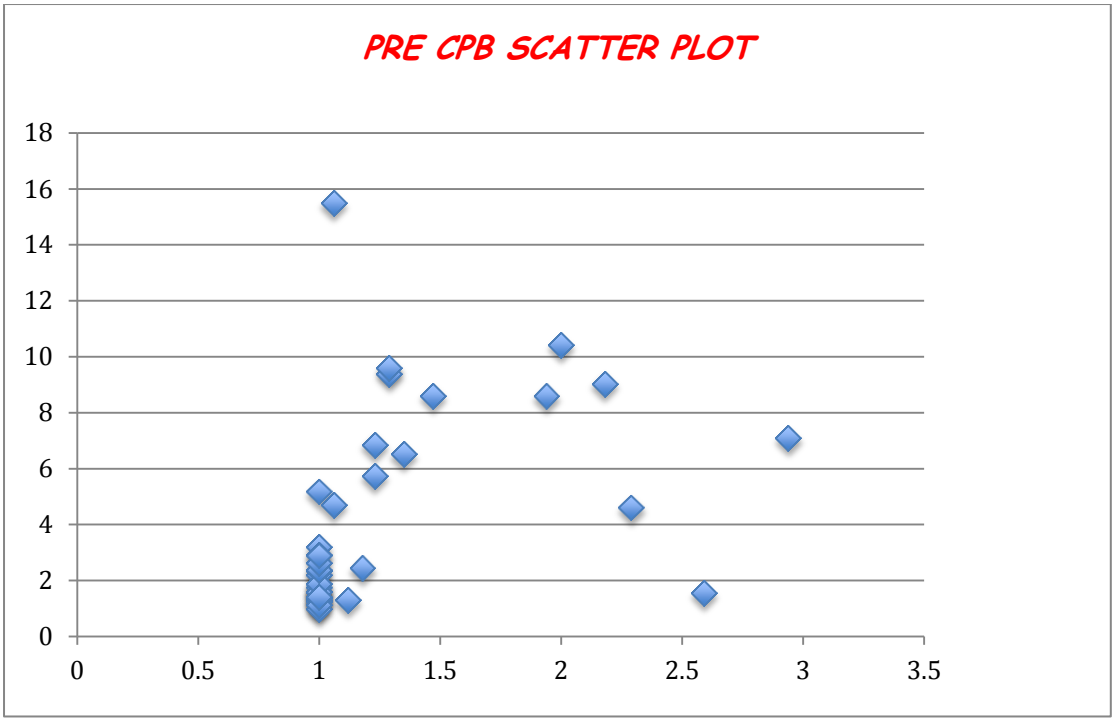


Figure 1: Pre CPB values of WMSI and SDI are plotted on x and y-axis respectively showing an average correlation between the two; Pearson correlation coefficient, r = 0.403; p value = 0.012.

Abbreviations:- *WMSI= Wall motion score index; SDI= Systolic dyssynchrony index.*

Post CPB identification of RWMA

Group A patients were divided into three subgroups (figure 2) on the basis of TEE observations noted before and after initiation of adrenaline.

Subgroup 1 [n =13] included patients who never developed RWMA (WMSI=1) at any time point with *SDI values falling in normal range* except in one patient. In that particular case, SDI crossed the upper limit of 5.6% despite of normal WMSI. In addition, we observed significant blue areas (figure 3) on average "Timing" polar map (bull's eye plot), which represented early activating segments (EAS).

In **Subgroup 2** [n=5], patients developed dyssynchrony (SDI>5.6%) before adrenaline that subsequently normalized with adrenaline (figure 4). They suffered transient segmental dysfunction, commonly involved areas being the apical segments as seen on Parametric Motion Imaging. But their WMSI values were reported normal in contrast to abnormal 3DE findings.

There were 4 patients in **Subgroup 3**, out of which 2 patients showed dyssynchrony only after adrenaline, unlike the former cases. Their SDI normalized after discontinuation of adrenaline (figure 5). In the third patient, new onset of LV dysynchrony (SDI>5.6%, WMSI=1) was determined after separation from CPB that did not resolve with adrenaline. The last patient of subgroup 3 experienced hypokinesia in the anterior wall and moderate mitral regurgitation (MR) due to compromised flow in venous graft. The routine 2D TEE evaluation detected hypokinetic anterior wall in this patient. After placement of additional venous graft in distal left anterior descending (LAD) artery, 2D echocardiography confirmed improvement in anterior wall contractility and grade of MR. However, RT3DE analysis showed disparity with 2D imaging as SDI was less than 5.6% at all time intervals indicating error in the 3D datasets processing.

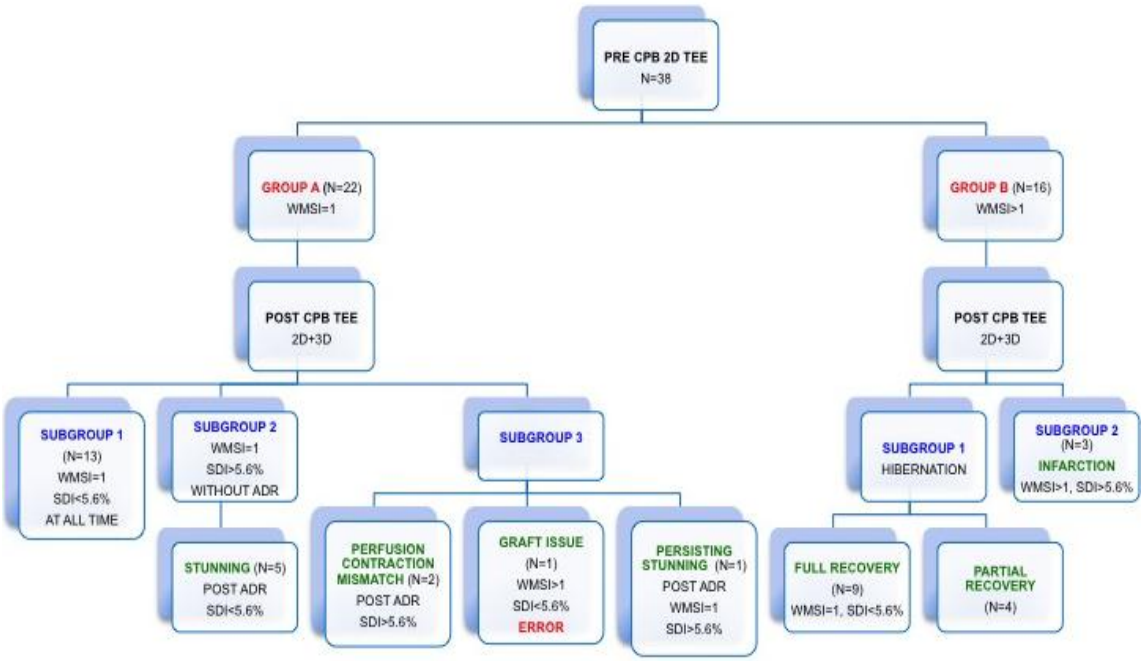


Figure 2: A flow chart showing division of patients in groups and subgroups based on pre and post cardiopulmonary bypass (CPB) transesophageal echocardiography (TEE) findings.

Abbreviations:- *WMSI= wall motion score index; 2D = 2 dimensional; 3D= 3 dimensional; SDI= systolic dyssynchrony index; ADR= adrenaline.*

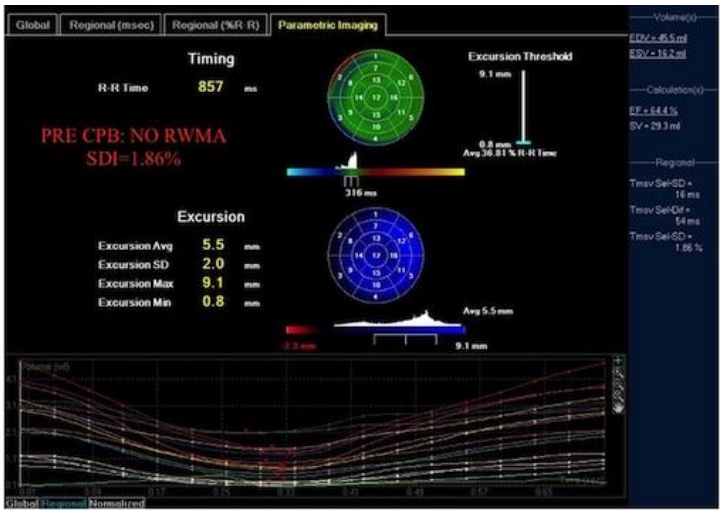


Figure 3A

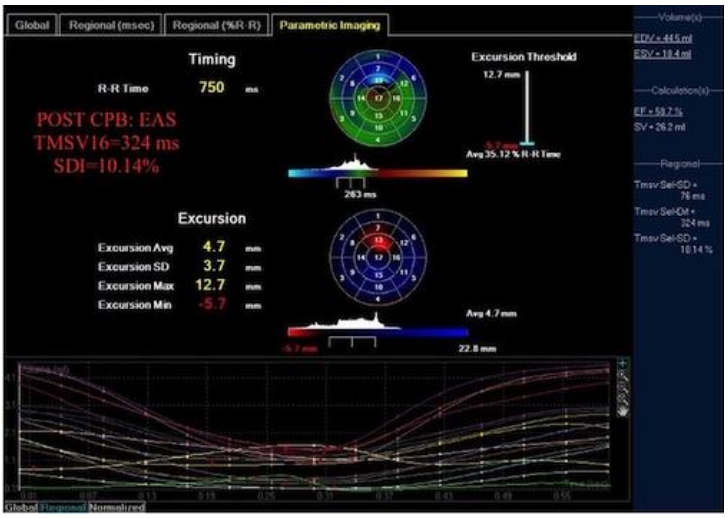


Figure 3B

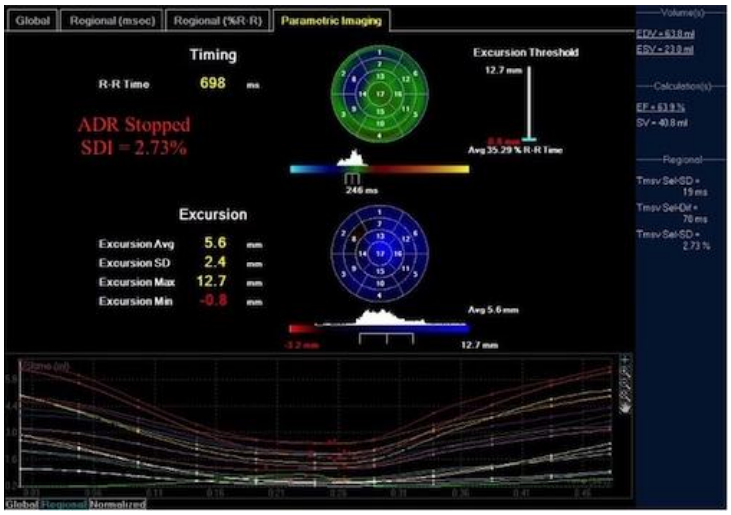


Figure 3C

Figure 3: Parametric bull's eye image showing ***EAS causing dyssynchrony that was resolved after stopping adrenaline.*** *3A: Pre CPB normal SDI; 3B: Post CPB* ***EAS (blue color on Timing plot)****, increased Tmsv16-diff, Tmsv16-SD, and SDI; 3C: Withdrawal of adrenaline improved mechanical dyssynchrony. EAS= early activating segments.*

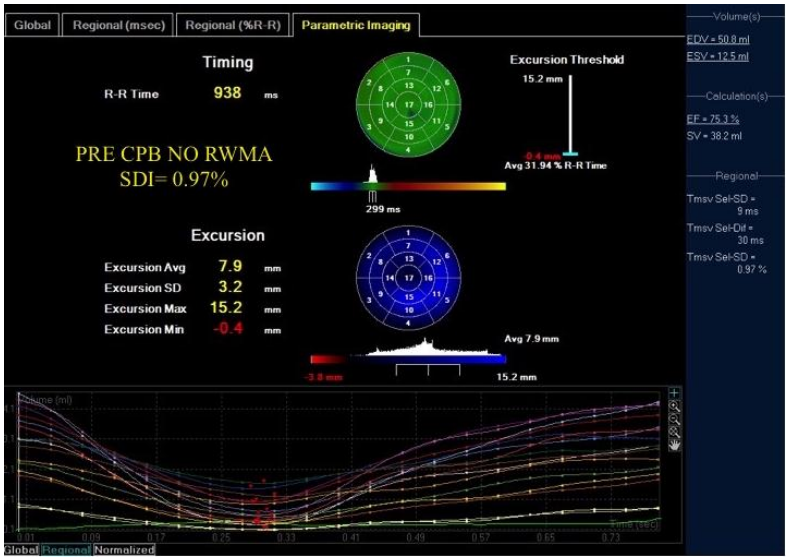


Figure 4A

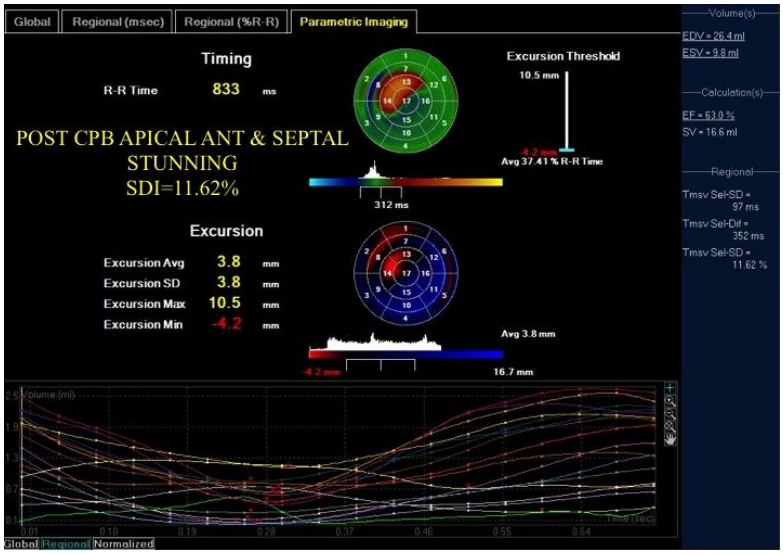


Figure 4B

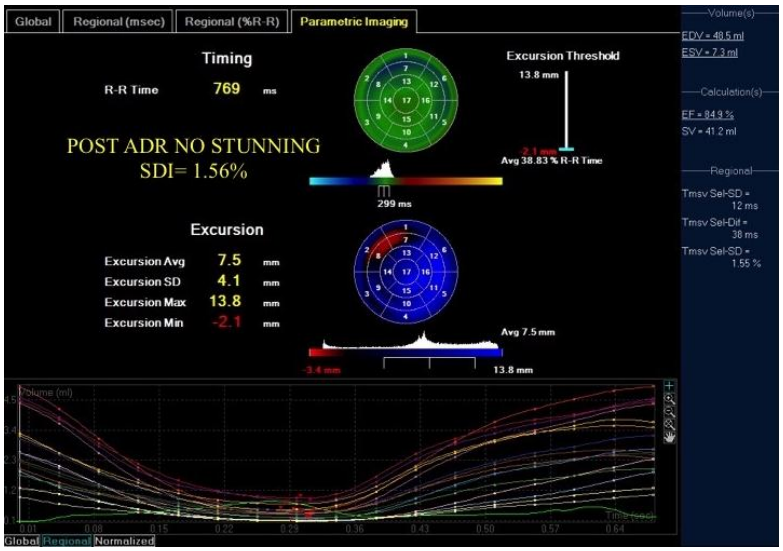


Figure 4C

Figure 4: RT3D parametric imaging demonstrating ***Stunned myocardium****. 4A: Pre CPB normal SDI; 4B: Post CPB* ***Stunning*** *in apical anterior and septal segments causing increased Tmsv16-diff, Tmsv16-SD, and SDI; 4C: Post ADR resolution of mechanical dyssynchrony.*

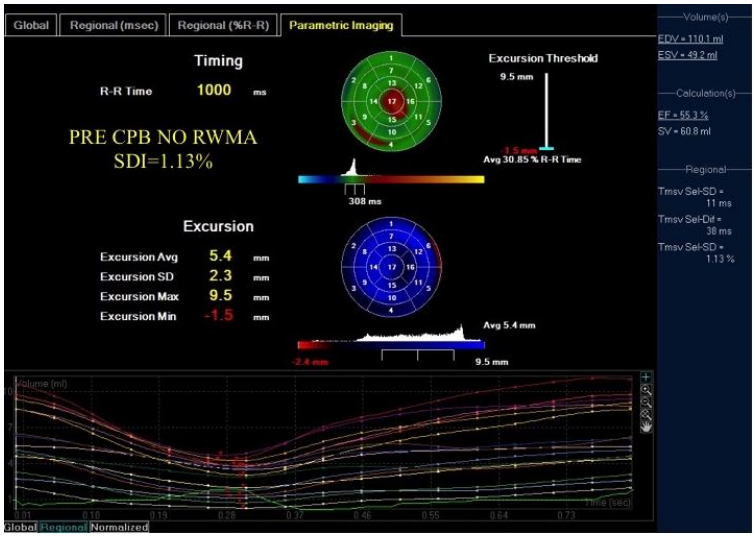


Figure 5A

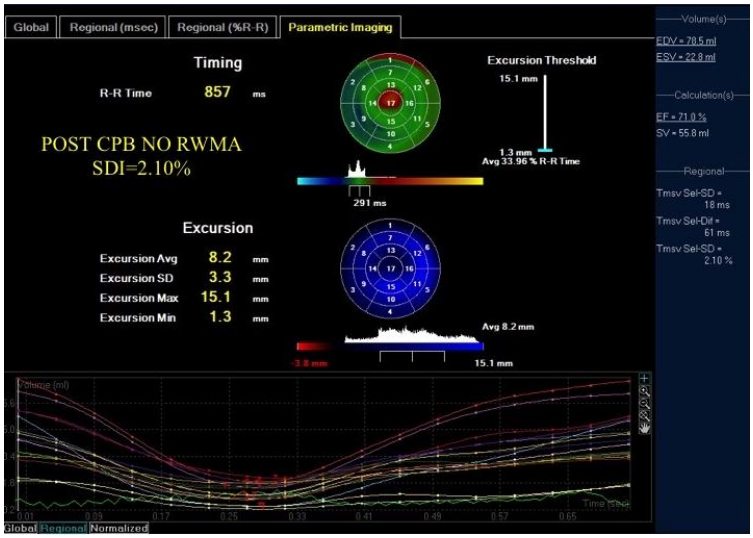


Figure 5B

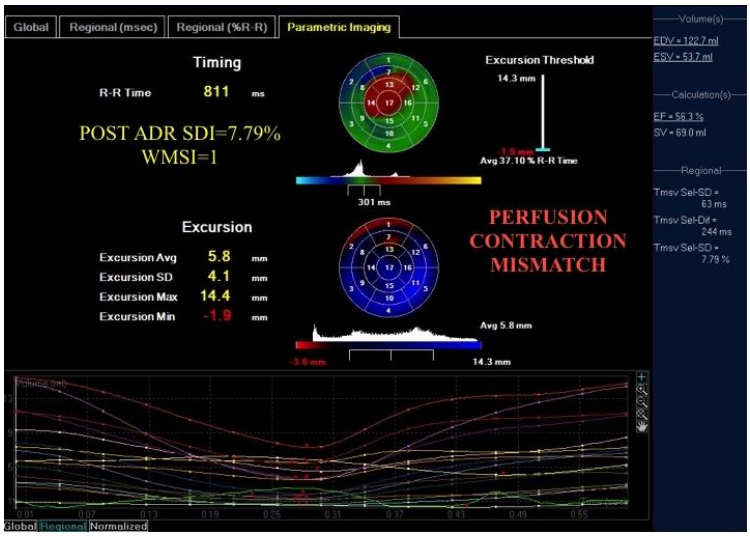


Figure 5C

Figure 5: RT3DE parametric imaging depicting ***Perfusion-contraction mismatch after adrenaline in a diabetic patient****. 5A and 5B: Pre and Post CPB normal SDI; 5C: Post ADR increase in SDI, red regions on Timing plot corresponding with black regions on Excursion plot.*

Similarly, inspection by TEE led to stratification of **Group B** patients into two subgroups (figure 2). **Subgroup 1** [n =13] included patients showing either full recovery (WMSI=1, SDI<5.6%; [n=9]) as shown in figure 6 or partial recovery of abnormal wall motion (WMSI>1, SDI>5.6%; [n=4]). Figure 7 revealed no improvement in myocardial contractility after revascularization in **Subgroup 2** patients (WMSI>1, SDI>5.6%; [n=3]). Another incidence of issue with the patency of venous graft culminating in acute ischemia presented with intra-ventricular dyssynchrony (figure 8), which later on improved after surgical revision.

The agreement between 2D and RT3DE assessment for identification of transient and persistent RWMA, before and after adrenaline infusion, were 76% (r=0.36; p=0.026) and 81% (r=0.68; p=0.001) respectively (table 3, figure 9).

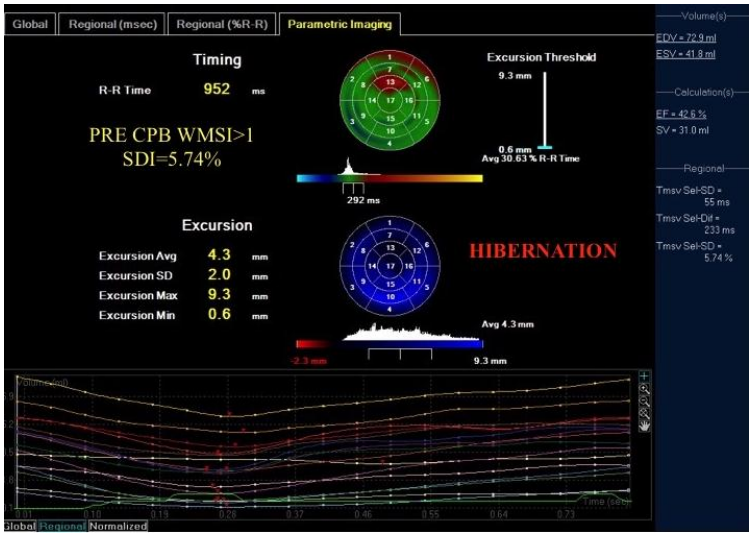


Figure 6A

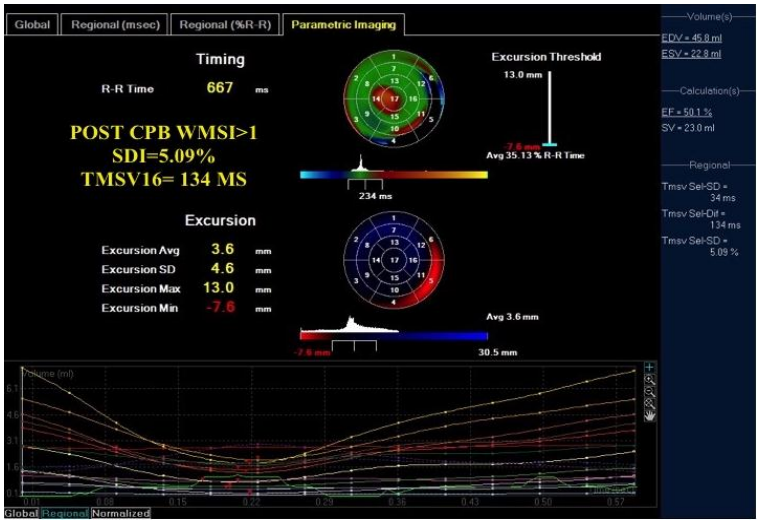


Figure 6B

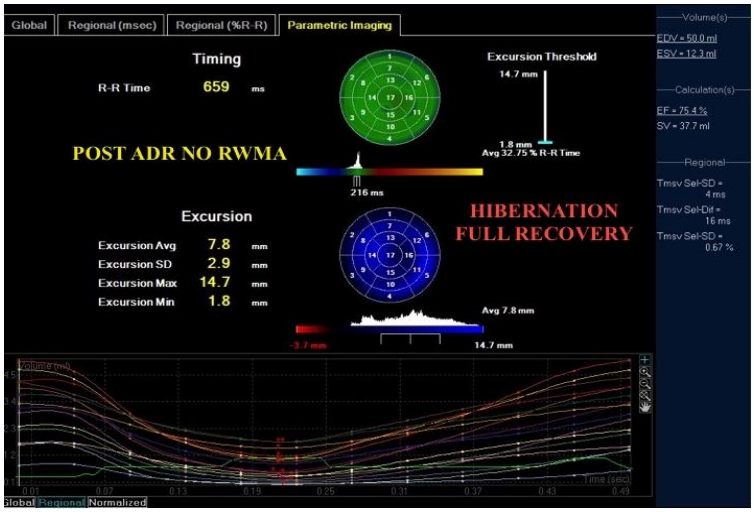


Figure 6C

Figure 6: RT3DE parametric imaging showing ***full recovery of hibernating segments*** *after revascularization. 6A: Pre CPB SDI=5.74%, WMSI>1; 6B: Post CPB SDI =5.09%, WMSI>1; 6C: Post ADR SDI=0.67%, WMSI=1.*

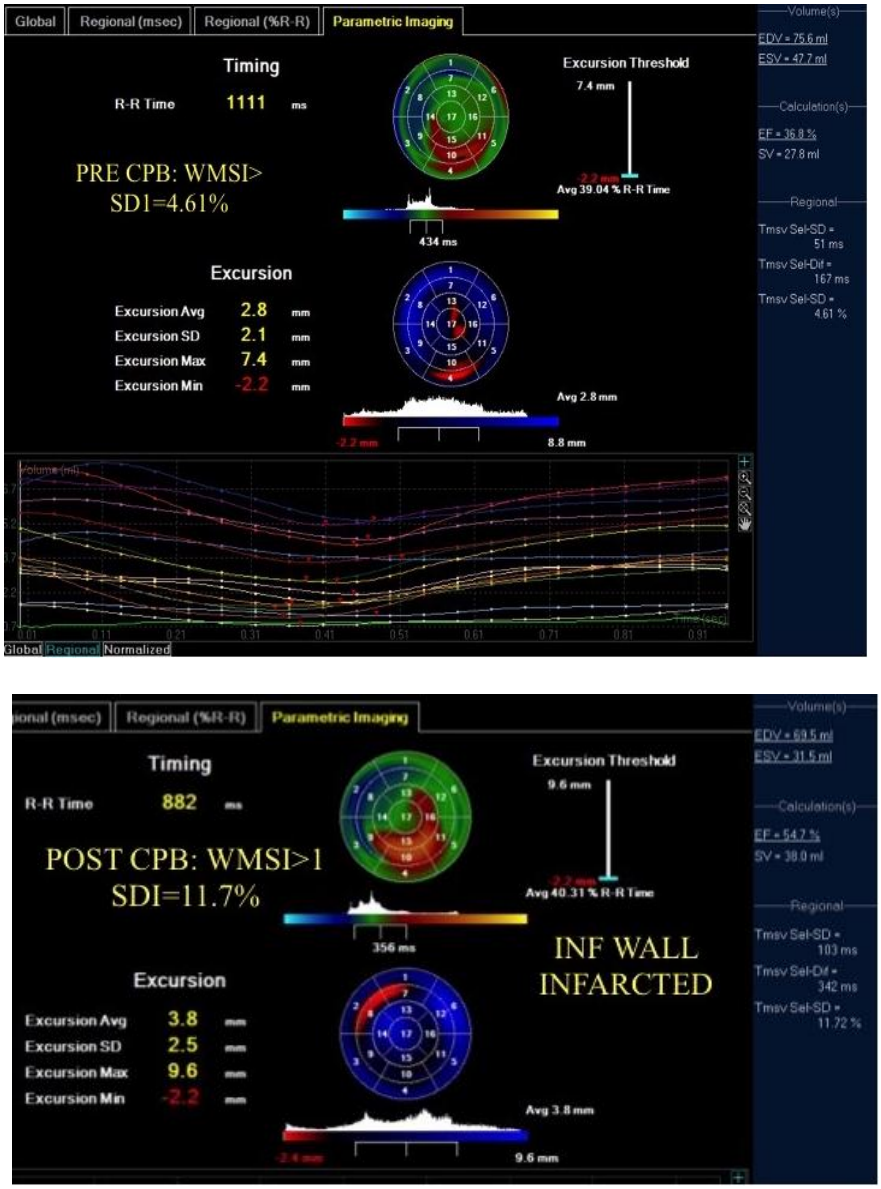


Figure 7A & 7B

Figure 7: RT3DE parametric imaging showing ***no recovery of infarcted segments*** *after revascularization. 7A: Pre CPB red areas on bull's eye plots identifying RWMA in inferior and inferolateral segments; 7B: Post CPB no improvement in SDI.*

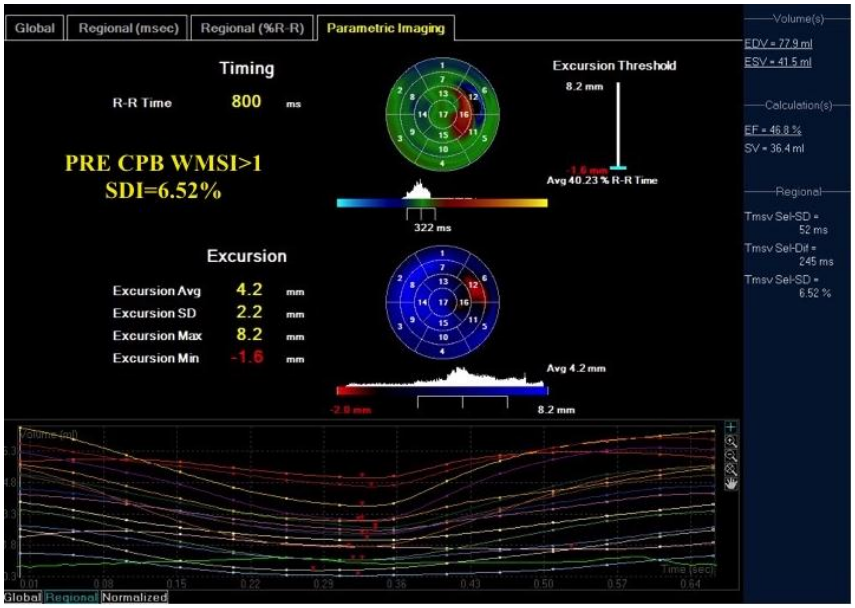


Figure 8A

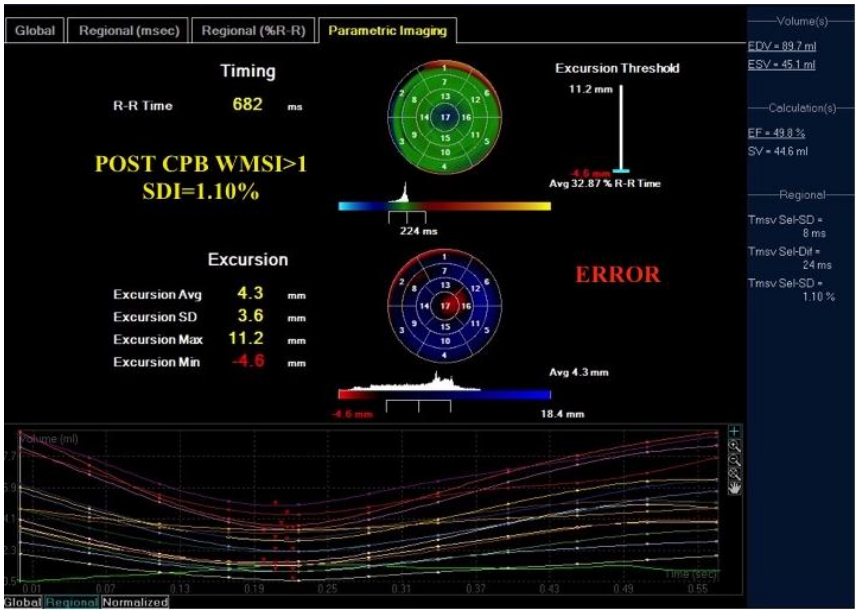


Figure 8B

Figure 8: RT3DE parametric imaging. 8A: Pre CPB SDI=6.52%, WMSI>1; 8B: Post CPB SDI =1.1%, WMSI>1 indicating ***failure of RT3DE to detect RWMA*** *due to graft dysfunction.*

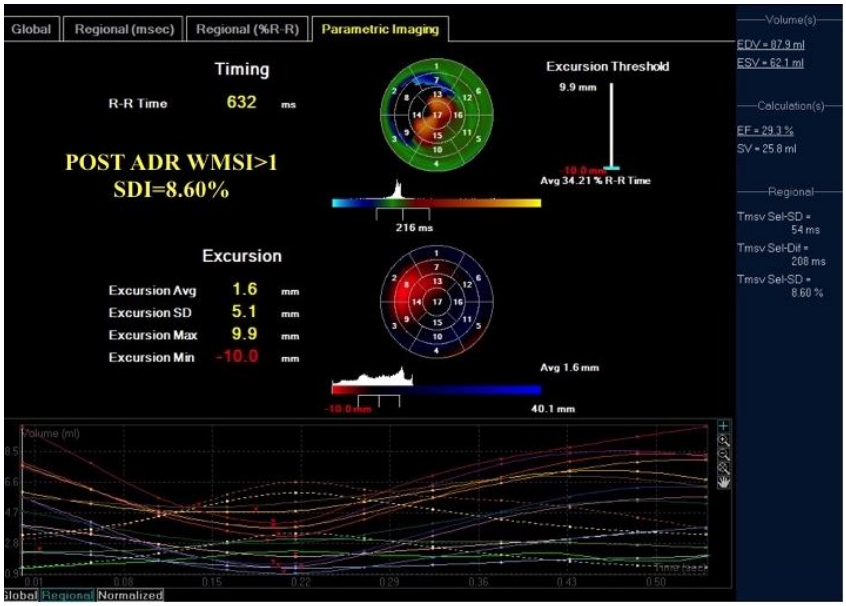


Figure 8C

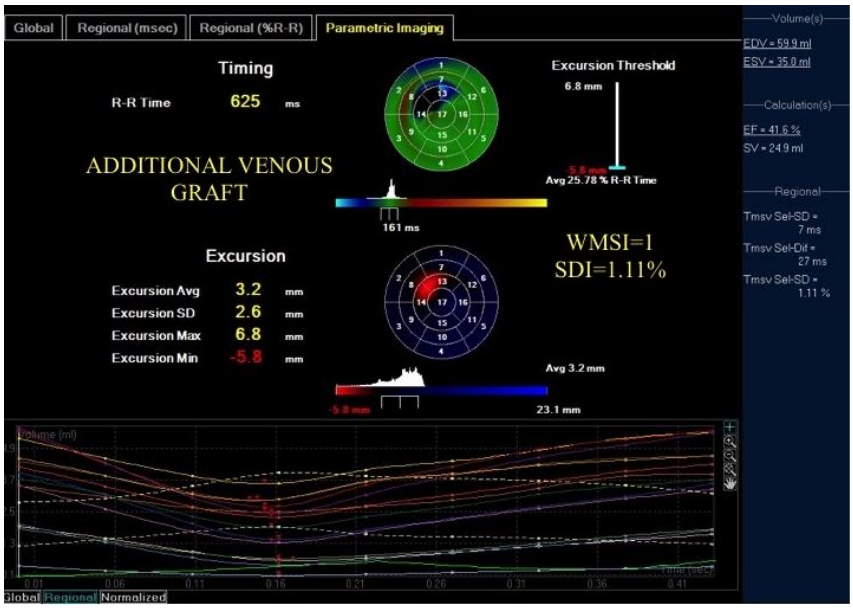


Figure 8D

Figure 8: RT3DE parametric imaging showing dyskinesia in anteroseptal and apical amterior segments due to ***Graft dysfunction.*** *8C: Post ADR SDI=8.6%, Tmsv16-diff= 208msec, Tmsv16-SD=54 msec, WMSI>1 due to graft dysfunction; 8D: Post graft revision TEE analysis shows improvement in SDI and WMSI.*

Abbreviations:- ADR = adrenaline; SDI = systolic dyssynchrony index; WMSI= wall motion score index.

POST CPB WITHOUT ADR **POST CPB WITH ADR**

VARIABLE (N=38)	**FREQUENCY**	**PERCENTAGE (%)**	**VARIABLE (N=36)**	**FREQUENCY**	**PERCENTAGE (%)**
WMSI > 1	11	28.95	WMSI > 1	9	25
SDI > 5.6%	18	47.37	SDI > 5.6%	10	27.78
WMSI >1 SDI >5.6% PERSISTING RWMA	9	23.68	WMSI>1 SDI>5.6% PERSISTING RWMA GRAFT ISSUE	5 4 1	13.89
WMSI = 1 SDI> 5.6% (EAS)	2	5.26	WMSI=1 SDI >5.6% (EAS)	2	5.55
2D = 3D	29	76.31	2D = 3D	29	80.55
2D ≠ 3D	9	23.68	2D ≠ 3D	7	19.44
WMSI =1 SDI> 5.6% (RED AREAS)	7	18.42	WMSI=1 SDI>5.6% (RED AREAS) TRANSIENT RWMA	3	8.33
RT3D ERROR WMSI>1 SDI <5.6%	2	5.26	WMSI >1 SDI <5.6%	4	11.11

Table 3 ***Level of agreement between WMSI and SDI after weaning from CPB*** *in numbers and percentages. Abbreviations:- WMSI, wall motion score index; SDI, systolic dyssynchrony index; CPB, cardiopulmonary bypass, 2D=3D, concordance between WMSI and SDI; 2D≠3D, discordance between WMSI and SDI.*

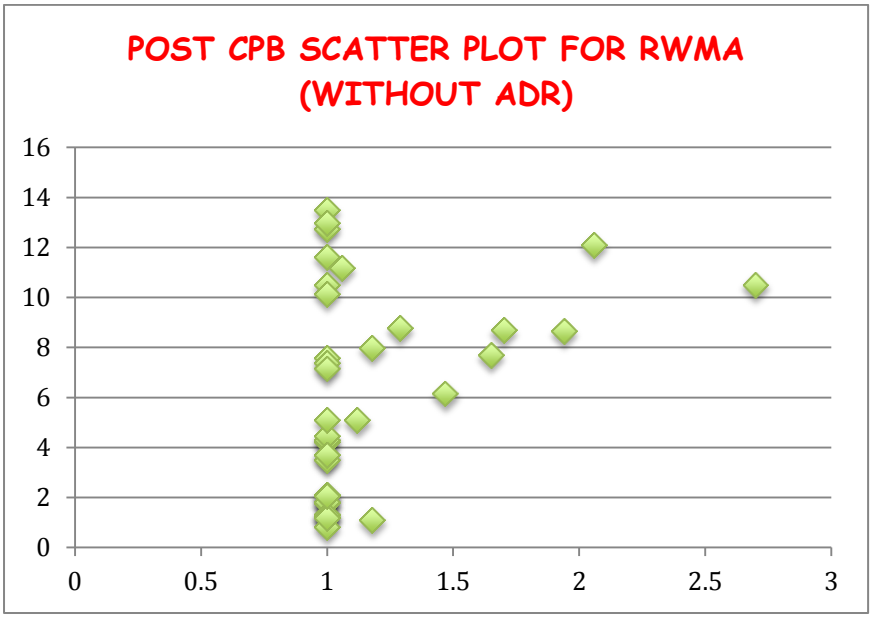


Figure 9A

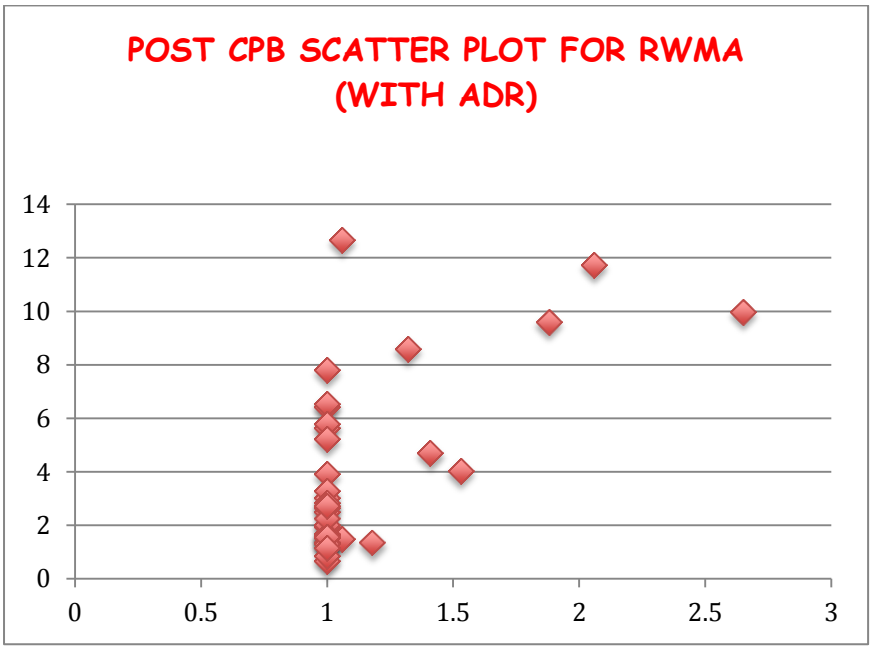


Figure 9B

Figure 9: Post CPB values of WMSI and SDI are plotted on x and y-axis respectively showing correlation between the two indices. *9A: Post-CPB Pearson correlation coefficient (r) of 0.36 without adrenaline; 9B: Post-CPB Pearson correlation coefficient (r) of 0.68 with adrenaline.*

RT3DE Quantification of RWMA

Table 4 shows the mean values of Tmsv 16-diff, Tmsv 16-SD, SDI and Exc avg observed during the operative phase in **Group A** patients. In subgroup 1 (Group A), pre-CPB and post-revascularization measured mean SDI values were 2.08±1.06 and 2.48±1.67 respectively, implying well synchronized segmental contractions. In addition, there was significant improvement in segmental excursion after revascularization (p=0.018). Patients who developed transient regional dysfunction immediately after weaning from CPB in subgroup 2 demonstrated significantly deranged values of Post CPB SDI (8.59±3.72; p=0.04) and Exc avg (4.5±0.7; p=0.04) but mechanical dyssynchrony gradually improved with adrenaline (SDI=2±0.79; p=0.04, Exc avg=6.12±2.13). The smallest cohort of 4 patients in subgroup 3 showed worsening in SDI values after adrenaline infusion (mean SDI, 1.72±0.8 [pre-CPB]; mean SDI, 3.82±2.5 [post-CPB]; mean SDI, 5.33±2.8 [post adrenaline]; p=0.17).

For subgroup 1 of **Group B**, the mean SDI values attained during three time intervals (pre-CPB, post-CPB without adrenaline and post-CPB with adrenaline) were 7.01±3.99, 6.93±3.56, and 4.09±3.38 respectively (table 5). The improvement in mechanical dyssynchrony was statistically significant after revascularization with inotropic support of adrenaline (p=0.03). Moreover, the corresponding mean values of WMSI for the specific time intervals were 1.45±0.45, 1.20±0.25 and 1.11±0.18. Hence, regional myocardial dysfunction as assessed by 2D wall motion scoring also showed significant improvement (p=0.009). RT3DE parameters could quantify RWMA in subgroup 2 also. In patients with persistent RWMAs, no significant change was noted in their mean SDI values (p=0.37) before and after the surgery.

The difference in average segmental excursion of patients in the two groups was statistically significant after revascularization (mean Exc avg, 5.42±1.41 [**Group A**]; mean Exc avg, 3.88±0.79 [**Group B**]; p=0.04).

Variable		SG 1	Paired t- test (P)	ANOVA (P)	SG 2	Paired t- test (P)	ANOVA (P)	SG 3	P value	Friedman (P)
Tmsv 16-diff Mean (SD)	Pre CPB	76.31 (49.25)		0.43	64.4 (29.1)		**0.02**	54.7 (12.4)		0.37
	Post CPB	104.92 (89.49)	0.37		**254.4 (93.7)**	**0.04**		101.5 (65.1)	0.14	
	Post ADR	70.36 (50.75)	0.28		58.6 (27.9)	**0.04**		**153.3 (90.3)**	0.27	
			0.76			0.89			0.14	
Tmsv 16- SD Mean (SD)	Pre CPB	20.23 (10.5)		0.39	18.2 (7.85)		**0.02**	15 (3.56)		0.47
	Post CPB	27.46 (21.72)	0.33		**68.8 (31.7)**	**0.04**		28.7 (18.5)	0.14	
	Post ADR	18.64 (14.24)	0.26		15.4 (5.2)	**0.04**		**36.75 (22.2)**	0.71	
			0.74			0.46			0.14	
Tmsv 16- SD % RR (SDI)	Pre CPB	2.08 (1.06)		0.30	1.93 (0.86)		**0.02**	1.72 (0.8)		0.17
	Post CPB	3.71 (1.89)	0.19		**8.59 (3.72)**	**0.04**		3.82 (2.5)	0.07	
	Post ADR	2.48 (1.67)	0.33		2 (0.79)	**0.04**		**5.33 (2.8)**	0.46	
			0.48			0.89			0.14	
Exc avg Mean (SD)	Pre CPB	4.95 (1)		0.28	7.12 (1.42)		0.07	5.43 (1.2)		0.81
	Post CPB	4.87 (1.26)	0.87		**4.5 (0.7)**	**0.04**		5.13 (2.2)	0.71	
	Post ADR	5.34 (1.02)	**0.018**		6.12 (2.13)	0.13		**4.8 (1.1)**	0.78	
			0.2			0.34			0.71	

Table 4: RT3DE quantification of regional wall motion in Group A patients

P value in subgroup 3 was calculated by Friedman's test followed by Wilcoxon signed rank test between two individual time points.

***Abbreviations**:- SG= subgroup; Tmsv 16-diff= maximal time difference to reach minimum systolic volume among 16 segments; Tmsv 16-SD = standard deviation of time to reach minimal systolic volume for 16 segments; CPB= cardiopulmonary bypass; ADR= adrenaline; SDI= systolic dyssynchrony index; Exc avg= average value of excursion deviation.*

Variable		SG 1	Paired t-test P value	ANOVA P value	SG 2	P value	Friedman P value
Tmsv 16-diff Mean (SD)	Pre CPB	248.6 (143.5)		**0.012**	203 (59.8)		0.60
	Post CPB	183.8 (95.2)	0.24		221 (84.1)	0.65	
	Post ADR	**93.8 (75.6)**	**0.01**		282.33 (70.9)	0.18	
			0.05			0.28	
Tmsv 16- SD Mean (SD)	Pre CPB	60.15 (39.9)		**0.035**	70 (26.9)		0.87
	Post CPB	46 (22.34)	0.30		81 (41.01)	0.65	
	Post ADR	**25.7 (22.4)**	**0.02**		77.5 (36.06)	0.31	
			0.02			0.60	
Tmsv 16- SD %RR (SDI)	Pre CPB	7.01 (3.99)		0.08	6.91 (2.21)		0.37
	Post CPB	6.93 (3.56)	0.96		10.41 (1.73)	0.28	
	Post ADR	**4.09 (3.38)**	**0.03**		10.43 (1.14)	1.0	
			0.06			0.11	
Exc avg Mean (SD)	Pre CPB	4.15 (1.14)		0.60	2.57 (0.4)		0.44
	Post CPB	4.34 (1.17)	0.61		3.1 (0.3)	0.18	
	Post ADR	4.64 (1.73)	0.64		3.13 (0.61)	1.0	
			0.41			0.28	

Table 5: RT3D TEE quantification of Regional wall motion in Group B patients

P value in subgroup 3 was calculated by Friedman's test followed by Wilcoxon signed rank test between two individual time points.

***Abbreviation**:- SG= subgroup; Tmsv 16-diff= maximal time difference to reach minimum systolic volume among 16 segments; Tmsv 16-SD= standard deviation in time to reach minimal systolic volume; CPB: cardiopulmonary bypass; ADR: adrenaline; SDI: systolic dyssynchrony index; Exc avg: average value of excursion deviation*

DISCUSSION

Discussion

Previous studies have reported various techniques of echocardiography to assess RWMA in patients undergoing CABG: 2D segmental wall motion analysis, segmental wall thickening [14], TDI and STI.[15] The commonly used method to quantitate regional myocardial dysfunction is by calculation of WMSI which has many limitations: 1) it is a subjective and semi-quantitative measure, subtle differences in wall motion are often missed visually, 2) it does not differentiate viable from the non viable tissue, 3) it may be influenced by an unmasked scar caused by changes in afterload and a tethering effect caused by neighboring infarcted segment[13]. It is a well-known fact that Doppler measurements suffer major drawback due to substantial angle dependency and TDI strain values may also get altered due to tethering effect. Though STI is the most reliable method to quantify RWMA with least probability of inducing errors in measurement of strain rate[15], the process is off-line and time consuming, thus limiting its use in acute ischemic events. Therefore, there is a lack of gold standard technique to assess regional LV dysfunction during the surgery.

RT3DE technology allows live imaging of the heart in the 3D domain, exploits the full volumetric information without relying on geometric modeling, and thus provides objective quantification of LV function.[16] It has shown a considerable promise in deriving intra-ventricular dyssynchrony and in the accurate selection of patients eligible for CRT.[6,7,9] Segmental dyssynchrony may occur either due to delayed activation of segments or due to abnormal contraction of segments (hypo/akinesis). In this context, Delgado V et al [9] found that among the patients of acute MI, the most delayed segments were more frequently located at the apical and mid segments corresponding with the areas of hypokinesia or akinesia. The study concluded that increased segmental dyssynchrony reflected higher degree of ischemic contractile

dysfunction. In addition, prevalence of LV dyssynchrony ranges from 20.8% to 79.6% in patients having systolic heart failure due to CAD, indicating myocardial ischemia being the harbinger of LV dyssynchrony. [8]

The present study has added more evidence to the clinical utility of RT3DE derived dyssynchrony and parametric imaging to quantitatively assess regional wall motion. This is the first study to examine the technique for identification and quantification of transient and persistent RWMAs in patients with ischemic cardiomyopathy undergoing myocardial revascularization procedure. We excluded patients with rhythmic disorders as it may also lead to intra-ventricular dyssynchrony, and thereby, SDI>5.6% determined contractile dysfunction exclusively, before and after revascularization.

RWMA assessment before Revascularization

The authors observed significant increase in mean values of SDI in patients with RWMAs (7.0±3.99) as compared to the patients with no RWMAs (1.91±0.9; p=0.01), the cut-off value used to define LV mechanical dyssynchrony was 5.6%.[6,26] In addition, patients with contractile dysfunction were found to have low values of average segmental excursion (Exc avg) and high values of negative excursion (figure 10). There was an inverse relation between SDI and LVEF, those with mechanical dyssynchrony were found to have depressed LV systolic function (mean EF, 63.4±6.1[group A]; 40.26±8.22[group B]). The results corroborated with the previous studies and strengthened the association of intra-ventricular dyssynchrony, abnormal regional contractility and systolic dysfunction in ischemic cardiomyopathy.[8,9] To define dyssynchrony, a moderate level of agreement has been studied earlier between SDI and TDI in different clinical scenario.[9,21,22] The present study tested the

concordance between SDI and WMSI, which was in line with the previous observations.

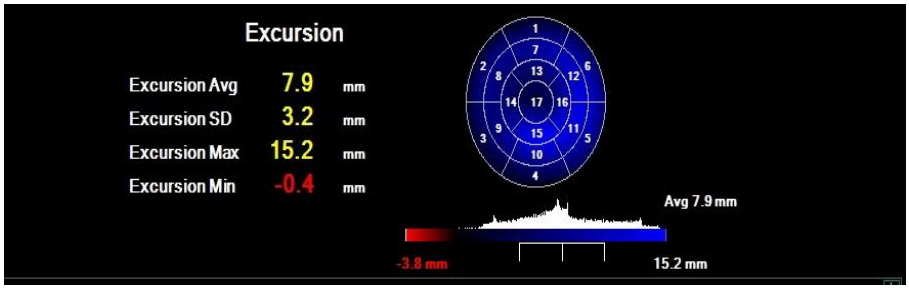


Figure 10A: Parametric Bull's eye image of a patient with no RWMA showing average excursion of 7.9 and negative systolic excursion of 0.4

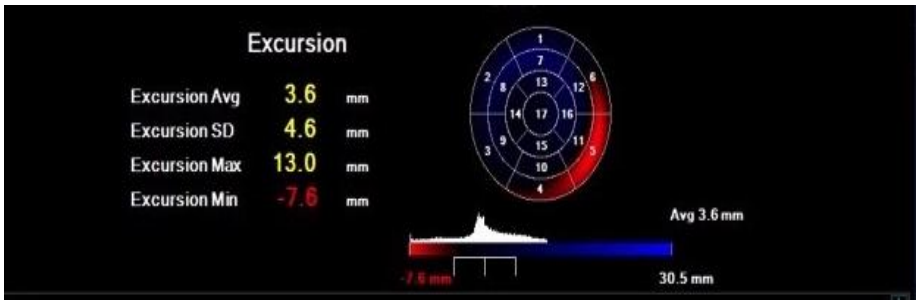


Figure 10B : Parametric Bull's eye image of a patient with RWMA showing average excursion of 3.6 and negative systolic excursion of 7.6

Pre-CPB discordance between SDI and WMSI:

Although there was a moderate degree of correlation between SDI and WMSI (r=0.4; p=0.012) for identification of RWMA during Pre-CPB period, the agreement level would be high if results of parametric motion imaging were included in correlation analysis. In 5 patients showing disagreement between the two techniques (WMSI>1; SDI<5.6%), there were noteworthy abnormalities in regional V-t curves (figure 11), values of Tmsv16-diff, Tmsv16-SD, Exc avg and bull's eye imaging. Hypokinetic and akinetic segments showed flattening of regional V-t curve while dyskinetic segments reached above 100% of EDV threshold on normalized V-t curve. Moreover, they had high negative values for systolic excursion (Exc min). These red regions of negative excursion on "Excursion plot" overlapped with red regions in the "Timing plot" which represented hypokinetic, akinetic or dyskinetic regions. One interesting finding was the presence of blue colored segments adjacent to red regions in "Timing plot" (figure 12 A). In patients with left bundle branch block (LBBB), there is mention of blue colored segments, which represent early activating segments (EAS) due to early activation of septal wall.[19] In the current study, the presence of EAS was the unusual finding after excluding patients with electrical abnormality. The authors suggest that hypercontractility of the adjacent segments trying to compensate for hypokinetic or akinetic regions [13] would have accounted for these blue colored segments. The maximal time difference to reach MSV would be between early activating segments and the most delayed segments and hence the values of Tmsv 16-diff were increased in these 5 patients. The algorithm calculated root mean square (RMS) value of timings in 16 segments to generate values of Tmsv 16-SD, which were further corrected to RR interval to compute SDI. The SDI values were normal as the RMS values of all the segmental timings including EAS and delayed segments would be corrected for

RR interval. Hence, SDI did not reveal dyssynchrony even in the presence of segmental wall abnormality. These hyperdynamic segments need to be differentiated from dyskinetic segments as curves for both are above threshold value of 100% of EDV and both are coded by red color on "Excursion plot" (figure 12B). Characteristic outward motion (paradoxical) during systole on 2D imaging and corresponding red colored areas on "Timing bull's eye plot" should be ascertained to call a segment dyskinetic whereas, a hypercontractile segment will show inward systolic motion in conjunction with blue areas on parametric "Timing plot". Hence, it is very essential to register abnormalities in polar maps even in the presence of normal SDI as endocardial wall motion is assessed more precisely with bull's eye imaging.[19] Therefore, in these 5 patients also, contractile dysfunction of myocardium could be determined by a thorough 3DE analysis, correlating well with 2D imaging. There was discordance in two patients where 2D and 3DE identification of abnormal segments did not match with each other.

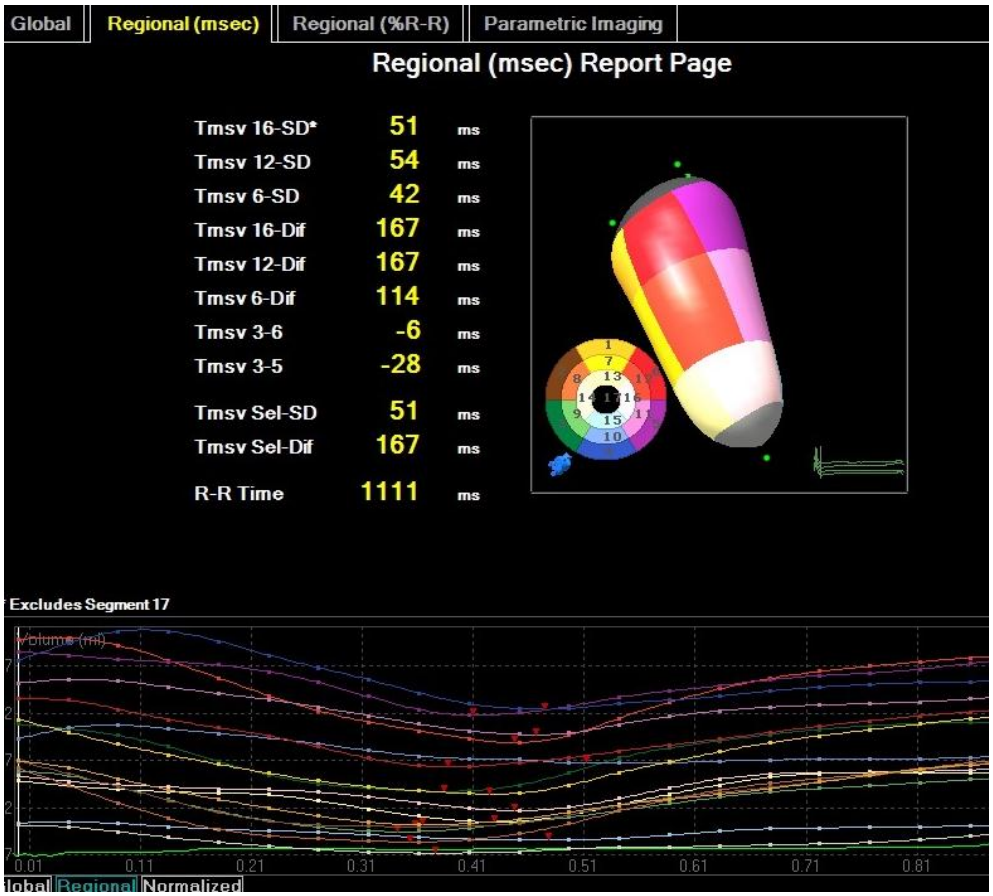


Figure 11A

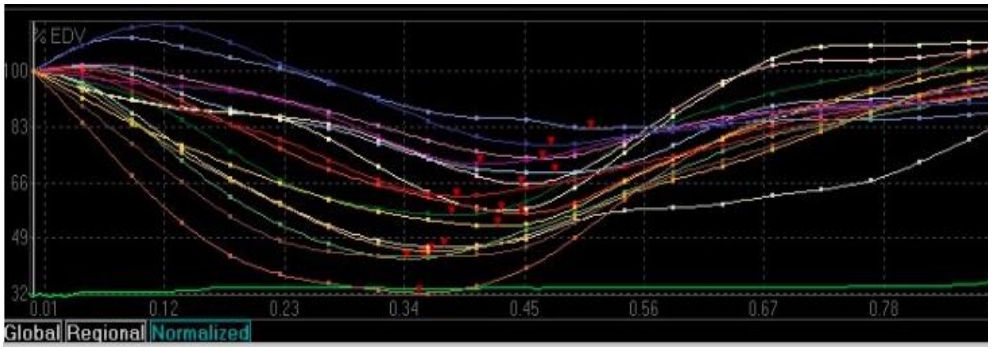


Figure 11B

Figure 11: Abnormal Volume-time (V-t) curves for hypokinetic and dyskinetic inferior wall segments showing Tmsv16-diff= 167ms and Tmsv 16-SD= 51ms in presence of normal SDI= 4.61%. *11A: Flattened regional V-t curve for inferior segments. 11B: Normalized V-t curve reaching above 100% EDV for basal and mid inferior segments.*

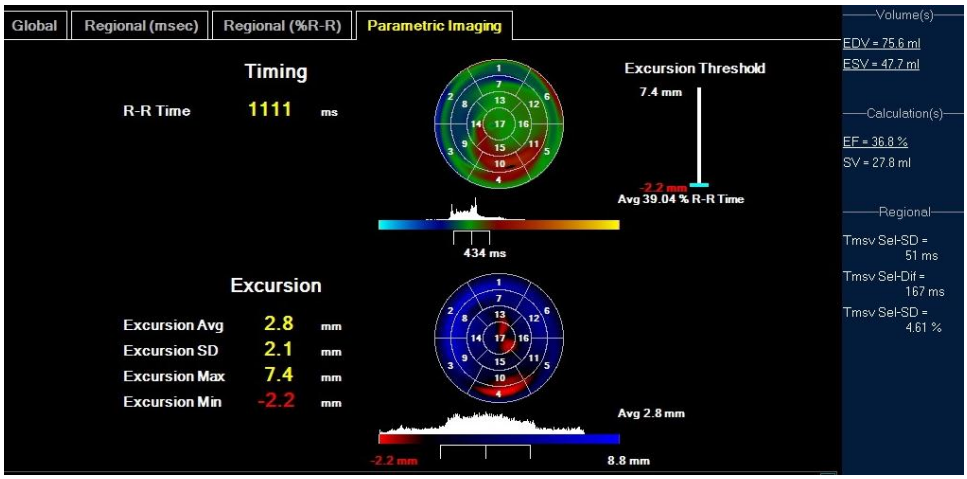
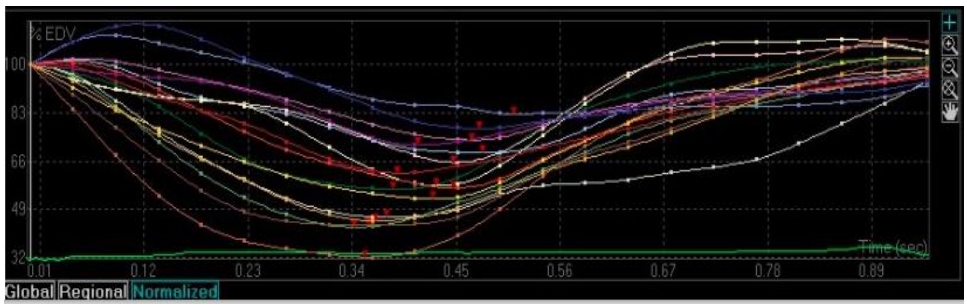


Figure 12A



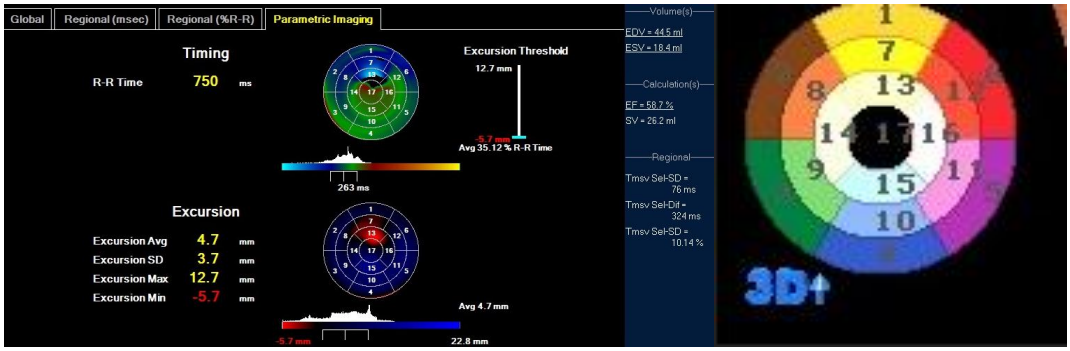


Figure 12B

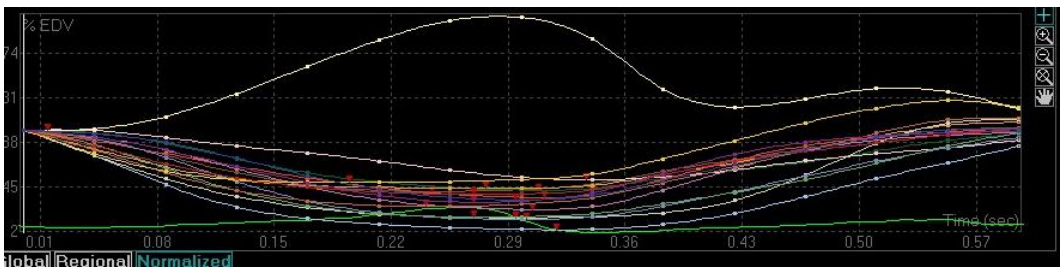


Figure 12: RT3DE analysis. *12A: Abnormal areas (basal inferior segment) on "Excursion Plot" generated dyskinetic V-t curve reaching above 100% EDV, Corresponding red areas are seen on "Timing plot"; 12B: V-t curve of red colored apical anterior segment seen on "Excursion plot" reaching above 100% EDV, Corresponding segment is seen in blue color on "Timing plot"indicating hyperkinesia*

RWMA assessment after Revascularization

In our study, RT3DE demonstrated normalization of SDI and improved systolic excursion after adrenaline infusion in patients who developed transient systolic dyssynchrony due to stunning after CPB. Patients identified with persistent RWMAs before revascularization, who demonstrated complete or partial restoration of ventricular synchrony on RT3DE were having hibernating myocardium, whereas, patients with scarred or infarcted myocardium showed persistent dyssynchrony. Therefore, it was possible to differentiate between viable and non-viable myocardium by RT3DE in post bypass period, which could be valuable in predicting functional recovery after revascularization. During surgery, 5 patients suffered acute ischemia and developed new transient regional dysfunction, 2 of them due to venous graft dysfunction and the other 3 due to perfusion-contraction uncoupling. RT3DE successfully quantified regional contractile dysfunction in four of them. It could also detect subsequent wall motion recovery after appropriate interventions by analyzing SDI values on regional V-t curves and color changes on bull's eye images.

Discordance between SDI and WMSI after revascualrisation

a) Without adrenaline

Out of 11 patients who demonstrated RWMAs on 2D echocardiography, mechanical dyssynchrony was noted in 10 of them. RT3DE failed to detect contractile dysfunction in one patient when he developed sudden apical anterior segment dyskinesia and mitral regurgitation. However, in the same patient, apical anterior segment ischemia could be identified later on by worsened SDI value at the third time point (after adrenaline infusion) as shown in figure 8C. The authors accept the fact it may be difficult to acquire and process 3D FV loop with accuracy in an emergency

scenario of acute ischemia when the major focus of an anesthesiologist shifts to stabilize the hemodynamics. Subsequently, when an additional venous graft was anastomozed to distal LAD on beating heart, RT3DE demonstrated intra-ventricular synchrony with resolution of RWMA.

In 6 patients, even though WMSI was normal, their values for Tmsv 16-diff, Tmsv 16-SD and SDI exceeded the upper limit of 60 msec, 32 msec and 5.6% respectively. On "Excursion plot", apical anterior and septal segments were either red, dark blue or black in color (figure 4) corresponding with the red areas on "Timing bull's eye plot". These segments probably represented stunned myocardium because of transient ischemic injury after aortic cross clamping and cardioplegic arrest leading to subtle global wall motion abnormality, not visually appreciable by 2D imaging. Mechanical synchrony was achieved under the effect of adrenaline after utilizing inotropic reserve of stunned myocardium. WMSI is a highly subjective assessment and marked changes in wall motion are required for diagnosing ischemia. The authors advocate the routine use of RT3DE as a complementary tool to 2D WMSI, being a more sensitive modality for identification and quantification of transient regional dysfunction due to stunning. Hence, the weak correlation between the two techniques ($r=0.36$; $p=0.026$) observed at this time period was due to better evaluation of stunned myocardium by RT3DE.

Another interesting finding on Parametric Motion Imaging was the appearance of EAS (blue color) on "Timing plot" in absence of adjacent red segments coherent with the red areas on "Excursion plot", believed to be due to hypercontractility (figure 3). SDI values in such cases were increased (>5.6%) with no abnormal wall motion on 2D imaging. Endogenous catecholamines are generally high after separation from CPB, and this may occasionally cause otherwise normally functioning segments to behave hyperdynamically [13] causing intra-ventricular dyssynchrony. The authors

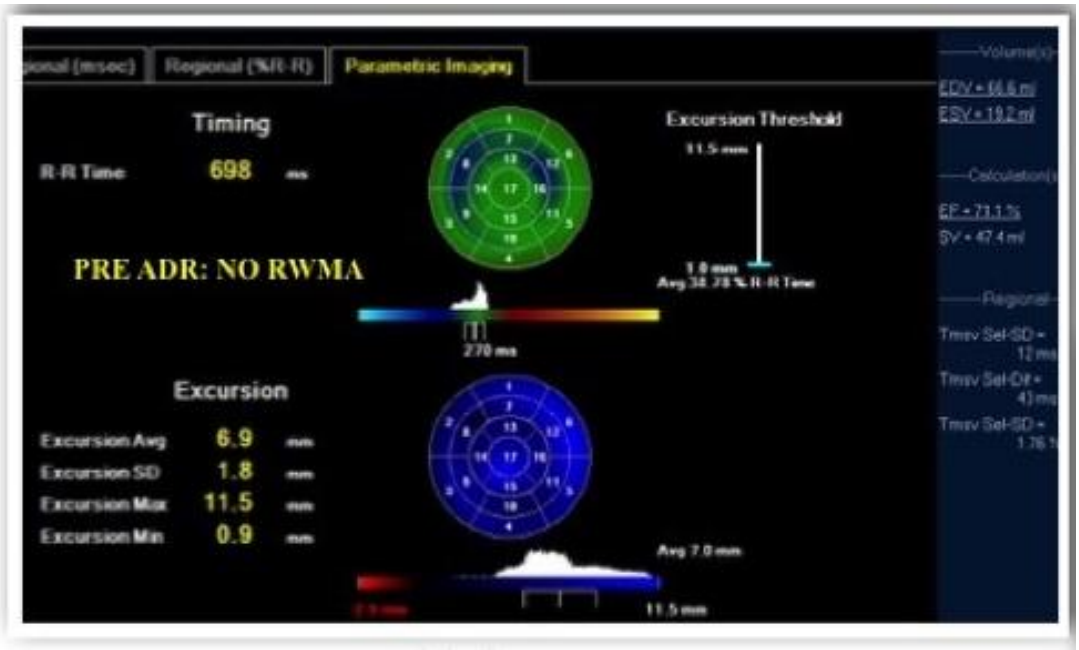
experienced that hypercontractility of segments could be potentially harmful, if at the expense of metabolic recovery. It led to perfusion-contraction mismatch and deterioration of tissue contractility in one case (figure 5).

b) With adrenaline

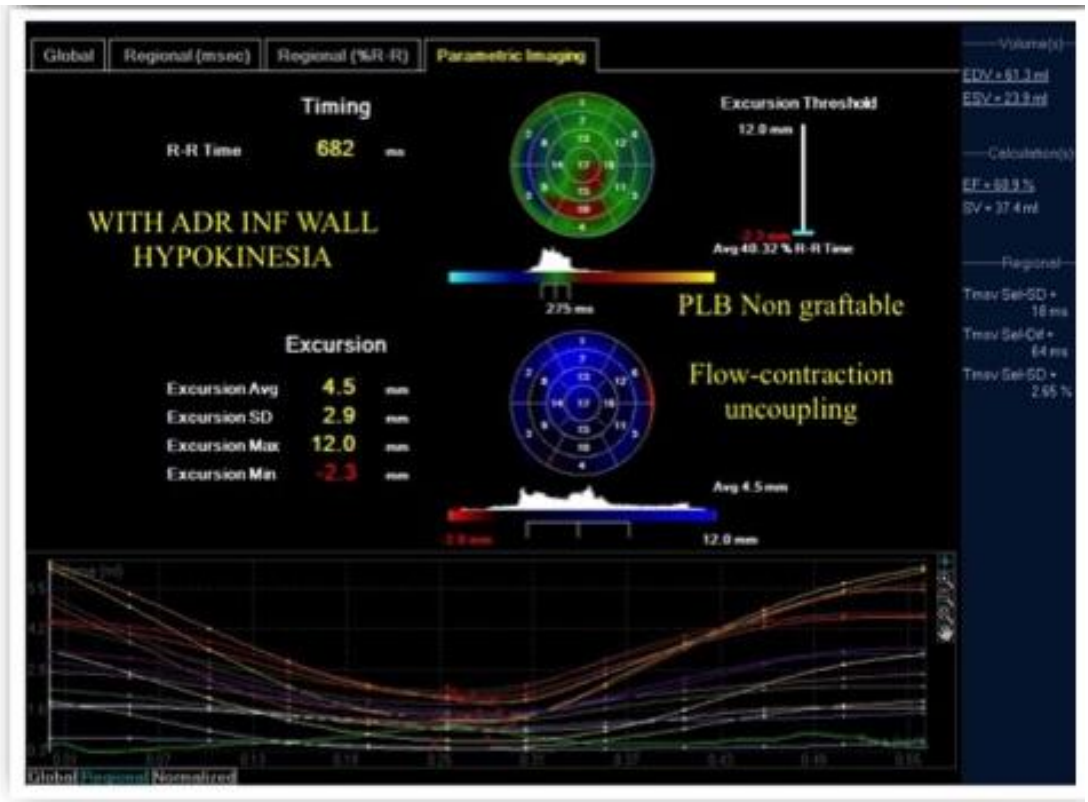
We observed an average correlation (r=0.68; p=0.001) between the two techniques employed for regional wall motion assessment after adrenaline. On inspection by 2DE, one patient showed inferior wall hypokinesia after adrenaline infusion with normal dyssynchrony index. Abnormal colored red areas were present on "Excursion plot" and "Timing plot", depicting ischemia in mid inferior wall territory (Figure 13). The operating surgeon found diffusely diseased posterior lateral branch of right coronary artery that was non-graftable. Flow- metabolism uncoupling in these less perfused areas under effect of adrenaline would have led to worsening of contractility. Adjacent myocardial segments started contracting more to compensate as evidenced by blue areas surrounding red areas on the "Timing plot" resulting in normal SDI and discordance with WMSI. This response further strengthened our idea of identifying blue segments on "Timing plot" as hypercontractile segments, which may create confusions and errors in RT3D assessment when an echocardiographer is just following SDI values. The new onset of RWMA in the inferior wall recovered after adrenaline was switched off. The same was confirmed by parametric motion imaging that also demonstrated increase in positive systolic excursion value. Furthermore, 2 patients who belonged to subgroup 3 of **Group A**, showed abnormal color pattern on polar maps (red areas with minimal blue regions on "Timing plot") on initiating adrenaline but in contrast to the above patient, they had associated increase in SDI with normal WMSI (figure 5). Inotropic and pressor effects of adrenaline may derange the delicate balance of flow-metabolism and result in mechanical

dyssynchrony before causing any obvious RWMA. This may be more evident in diabetic patients who are more prone to possess diffusely diseased distal intramyocardial vessels. Segmental contractions were more uniform after withdrawal of adrenaline in such cases. Therefore, elective use of adrenaline may not be beneficial in all cases, as stable hemodynamics, normal WMSI and mechanical dyssynchrony, all may coexist and remain unrecognized. This salient observation is yet another added advantage of RT3D parametric imaging over 2D TEE utilizing which we can modify tissue inotropic state and preserve perfusion-contraction coupling.

As mentioned above, EAS (blue color) represented hypercontractile segments, but it is important to understand that they may arise spontaneously without the need to compensate for adjacent hypokinetic regions. In such scenario, there will be increment in dyssynchrony with adrenaline. Though adrenaline caused increased contractility in well-perfused areas, it occurred at the cost of worsening intraventricular dyssynchrony that may reduce the mechanical efficiency. Therefore, PMI provided more insight than 2D imaging for management of the patients as we could achieve more coordinated segmental contractions without subjecting them to unnecessary inotropy.

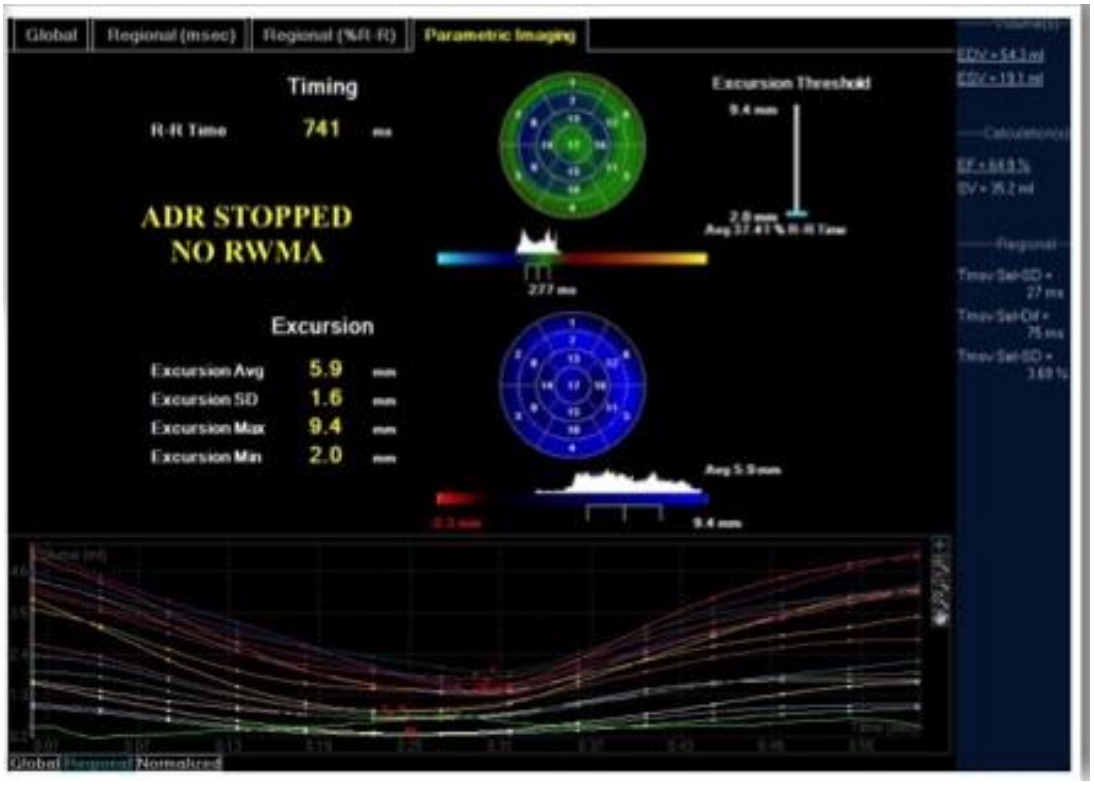


13 A



13 B

Figure 13: RT3D parametric imaging after weaning from CPB. *13A: Pre- ADR image showing no RWMA and SDI=1.76%; 13B: Post –ADR image showing inferior wall hypokinesia due to* ***Flow-metabolism uncoupling****, red color in mid-inferior segment on Timing plot and corresponding dark blue areas on Excursion plot with negative systolic excursion.*



13 C

Figure 13: RT3D parametric imaging after weaning from CPB. *13 C: RWMA resolved after withdrawal of adrenaline with simultaneous disappearance of abnormal colored areas on bull's eye. Excursion plot is displaying increase in positive systolic excursion values and no negative excursion.*

LIMITATIONS

Limitations

The study examined the feasibility of RT3DE to assess regional wall motion and compared it with WMSI. We have not used TDI and STI modalities for quantifying regional wall motion. Despite technological advances, RT3DE has relatively low temporal and spatial resolution and high dependence on image quality, reducing its feasibility in patients with dilated LV where border detection may become impossible. In addition, rhythmic cardiac disorders like atrial fibrillation may interfere with measurements and further limit the clinical applicability. The standard values of segmental excursion are not known to identify hypokinesia. Moreover, the study size was small, and a large prospective study is needed to validate the utility of SDI and parametric motion imaging. There is a need to explore normal and cut off values of maximal and minimal excursion. We did not perform reproducibility of RT3DE but previous studies have reported a low intra- and inter-observer variability for SDI measurement. [7,9]

CONCLUSION

Conclusion

Compared to 2D imaging, RT3DE parametric motion imaging enhances the ability to detect and quantify transient (acute) and persistent (chronic) ischemic contractile dysfunction in patients undergoing CABG. It thus provides a valuable complementary tool to routine 2D imaging. In the post cardiopulmonary bypass period, it guided us whether or not to administer inotropic support.

The results of current study can be summarized as:

1) RT3DE is a feasible modality for precise quantification of regional wall motion during revascularization procedure.

2) Systolic dyssynchrony index is a reliable measure to study the recruitment of viable segments after revascularization surgery and inotrope administration.

3) Improvement in mechanical dyssynchrony after revision of venous graft evaluates the efficiency of surgical intervention and patency of grafts.

4) The combined application of Parametric Motion Imaging and SDI improves the correlation with 2D segmental wall motion analysis.

5) PMI can detect acute transient regional dysfunction due to stunning, perfusion-contraction mismatch and graft dysfunction.

6) Bull's eye imaging has added advantage of recognizing hypercontractile segments (EAS) causing mechanical dyssynchrony.

7) RT3DE has potential to distinguish between viable and non-viable segments in the post cardiopulmonary bypass period and may predict functional recovery after revascularization.

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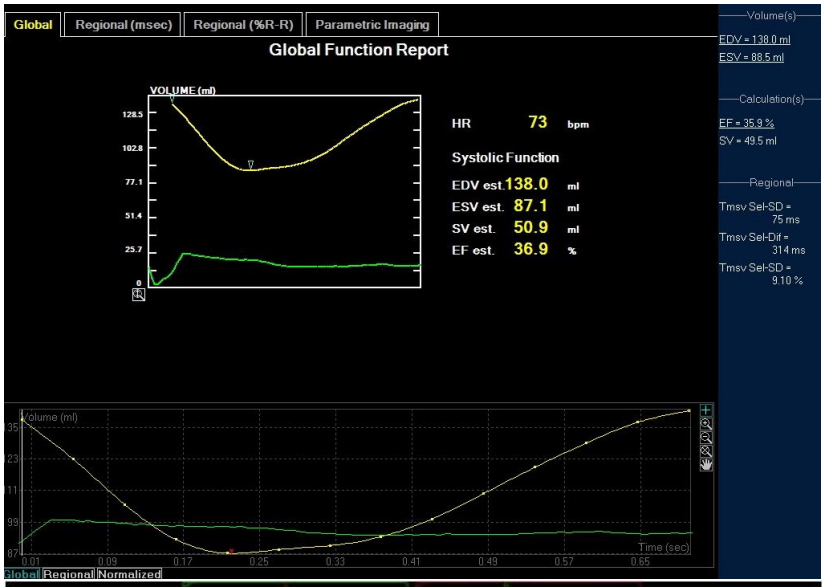
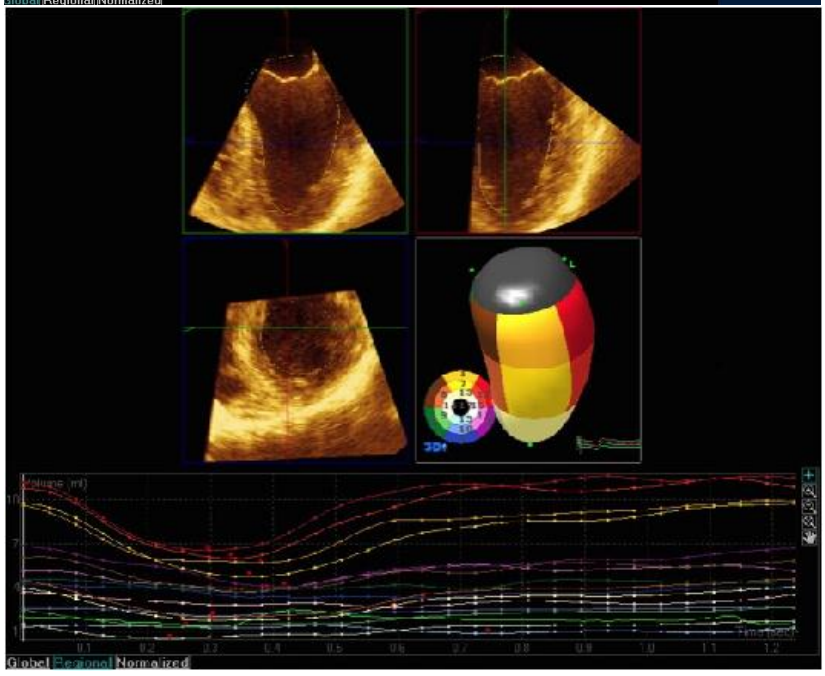
ANNEXURES

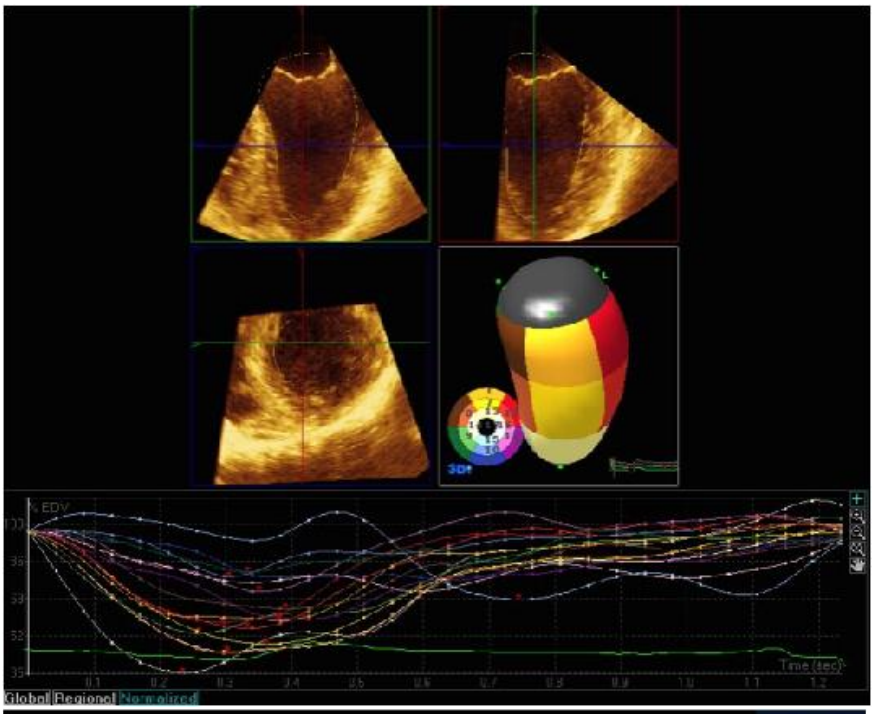
Annexure 1



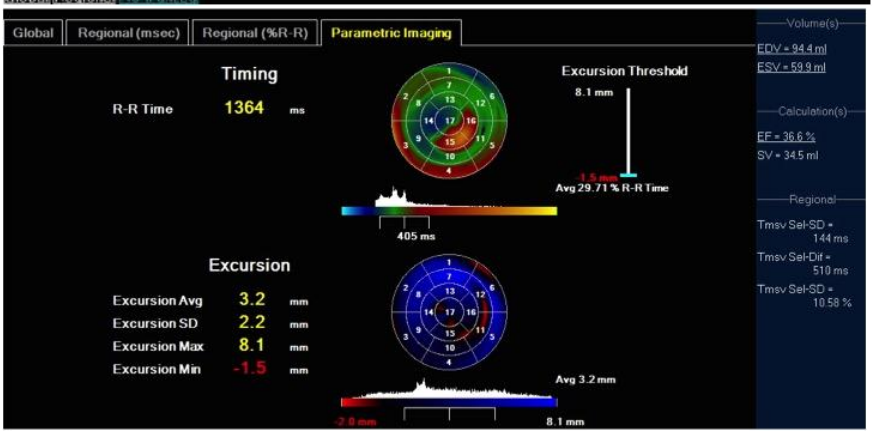
1A: Global Volume-time curve



1B: Regional Volume-time curve



1C: Normalized Volume-time curve



1D: Parametric Motion Imaging

Annexure 2

STUDY PROFORMA

Patient name-
Age/ sex-
Hospital no-
Pre operative TTE findings-

Diagnosis –
Height/ weight-
Plan of surgery-

Intraoperative TEE findings-

Variables		**Pre CPB**	**Post CPB (5 min) No inotropes**	**Post CPB With Adrenaline**
Transmitral flow velocity (m/s)	E velocity			
	A velocity			
	E/A			
	Adur (msec)			
	Vp			
Pulmonary vein velocity (cm/s)	S velocity			
	D velocity			
	S/D			
	AR velocity			
	AR dur			
MVannular velocity (m/s)	E’			
	E/E’			
	IVRT (msec)			
2D Imaging	EF			
	CO(L/min)			
	WMSI			
	RWMA			
3 D Imaging	EF			
	Tmsv16-diff			
	Tmsv12-diff			
	Tmsv 6-diff			
	Tmsv16-SD			
	SDI			
	Timing plot			
	Excursion plot			
	Exc Avg			

Intraoperative Surgical finding :

Any post CPB intervention :

Annexure 3

INFORMED CONSENT FORM

TITLE OF THE STUDY: "Intraoperative assessment of Transient and Persistent Regional left ventricular wall motion abnormalities in patients undergoing coronary revascularization surgery using Real time 3D Echocardiography"

Study number:
You are being requested to give consent to use the intraoperative transesophageal echocardiographic (TEE) findings during the coronary artery bypass surgery in our study. Intraoperative TEE is routinely performed as a standard of anaesthesia care in our hospital. This study will be conducted in 40 patients undergoing elective CABG.

What is the rationale of TEE assessment?
Intraoperative TEE is the standard care of monitoring that we provide to all patients undergoing any cardiac surgery unless otherwise contraindicated. In addition to confiming preoperative transthoracic echocardiographic findings, it helps in monitoring and guiding the intraoperative course. We will perform parametric imaging with the aim to assess intraoperative regional myocardial function.

Does intraoperative TEE have any side effects?
Insertion of probe may cause oral and gastric injury if inserted forcefully. We are routinely using this monitor and take adequate precautions to avoid injuries. We use good amount of lubricants and advance it gently without forcing it against resistance.

If you take part what will you have to do?
If you agree to include your observations in our study, no additional procedure/ intervention / follow up is required. All the data will be recorded in the operation theatre itself during routine surgery.

Can you withdraw from this study after it starts?
Your consent to use the collected data in this study is entirely voluntary. If you don't give this consent , this will not affect your usual treatment at this hospital in any way. Intraoperative TEE assessment will still be done as a part of standard anaesthesia care.

What will happen if you develop any study related injury?
We just want to use your data collected that we usually do during the routine surgery**. We donot expect any injury to happen to your patient during this study.**

If in case u develop any injury due to the procedure, it will be dealt as per the institute protocol.

Will you have to pay for the investigations?
The study will not add anything extra to the total cost of your surgery as it is an observational study with no added intervention from our side.

What happens after the study is over?
As we are not performing any additiona/new intervention, this study will not alter the course of surgery intraoperatively and postoperatively.

Will your personal details be kept confidential?
The results of this study will be published in a medical journal but you will not be
identified by name in any publication or presentation of results. However, your
medical notes may be reviewed by people associated with the study, without your
additional permission.
Your record number will not be used in analysis. Instead, a master key will be prepared linking it with the proforma serial number separately.

If you have any further questions, please ask
Dr Neelam Aggarwal (senior resident, department of cardiac anaesthesia), Telephone no. 9946551104, mail id- neelamdoc@sctimst.ac.in

Dr. Unnikrishnan (Additional Professor, department of cardiac anaesthesia)
Telephone no. 9446177521, email – unnikp@gmail.com

Declaration

Participant's name: Date of Birth / Age (in years):
I_____
_____,
son/daughter/mother/father of _____
(Please tick boxes) •
declare that I have read the above information provide to me regarding the study: **"Intraoperative assessment of Transient and Persistent Regional left ventricular wall motion abnormalities in patients undergoing coronary revascularization surgery using Real time 3D parametric motion imaging"** and have clarified any doubts that I had. [ ]
• I also understand that my participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my usual treatment or my legal rights [ ]
• I also understand that intraoperative TEE will be routinely performed anyways in our case. [ ]
• I also understand that the study is not posing any additional risk. It is an observational study. [ ]
• I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access [ ]
• I understand that my identity will not be revealed in any information released to third parties or published [ ]
• I voluntarily agree to take part in this study [ ]
• I received a copy of this signed consent form [ ]
Name:
Signature:
Date:
Name of witness:
Relation to participant:
Date:
(Person Obtaining Consent) I attest that the requirements for informed consent for
the medical research project described in this form have been satisfied. I have
discussed the research project with the participant and explained to him or her in
nontechnical terms all of the information contained in this informed consent form,
including any risks and adverse reactions that may reasonably be expected to
occur. I further certify that I encouraged the participant to ask questions and that
all questions asked were answered.

_____ _____
Name and Signature of Person Obtaining Consent

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान
तिरुवनन्तपुरम - 695 011, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY
THIRUVANANTHAPURAM - 695 011, INDIA
(An Institute of National importance under Govt. of India)

Institutional Ethics Committee

(IEC Regn No. ECR/189/Inst/KL/2013)

SCT/IEC/ 711/DECEMBER -2014 09-03-2015

Dr. Neelam Aggarwal
Senior Resident
Department of Cardiac Anaesthesiology
SCTIMST, Thiruvananthapuram

Dear Dr. Neelam Aggarwal,

The Institutional Ethics Committee reviewed and discussed your application to conduct the study entitled "INTRAOPERATIVE ASSESSMENT OF TRANSIENT AND PERSISTENT REGIONAL LEFT VENTRICULAR WALL MOTION ABNORMALITIES IN PATIENTS UNDERGOING CORONARY REVASCULARIZATION SURGERY USING REAL TIME 3D PARAMETRIC MOTION IMAGING" – A PROSPECTIVE OBSERVATIONAL STUDY" (IEC/711) on 20th December, 2014.

The following documents were reviewed:

Original submission

1. *Covering letter addressed to the Secretary, IEC, SCTIMST.*
2. *TAC Approval Letter.*
3. *IEC Application form.*
4. *TAC Application form.*
5. *Declaration.*
6. *Study proposal.*
7. *Informed consent form in English and Malayalam.*
8. *Observation chart.*
9. *Short CVs of PI and Co-PIs.*

Revised submission

10. *Covering letter addressed to the Secretary, IEC, SCTIMST dated 06.03.2015.*
11. *Modified Informed consent form in English and Malayalam*

तार : चित्रमेट फोन फाक्स ई-मेइल
Grams : Chitramet Phone : 2443152 Fax : (91)471-2446433 E-mail : sct. @sctimst.ker.nic.in
2550728

The following members of the Ethics Committee were present at the meeting held on 20th December, 2014 at G. Parthasarathi Board Room, AMCHSS, SCTIMST.

SL. No.	Member Name	Highest Degree	Gender	Scientific /Non Scientific	Affiliation with Institution(s)
1.	Justice Gopinathan. P.S	BSc. LLB	Male	Legal Expert (Chairperson)	No
2.	Dr. J. M. Tharakan	MD	Male	Clinician (Cardiologist)	Yes
3.	Shri. O.S. Neelakandan Nair	BE	Male	Engineer	Yes
4.	Dr. M.D. Gupte	MD, DPH	Male	Public Health	No
5.	Dr. Rema M. N	MD	Female	Pharmacologist	No
7.	Smt. Sathi Nair	MA	Female	Lay Person	No
8.	Dr. K R S Krishnan	ME, PhD	Male	Biomedical Scientist/Engineer	No
9.	Dr. Mala Ramanathan	MSc, PhD, MA	Female	Ethicist/Social Scientist (Member Secretary)	Yes

IEC Decision

The IEC approved the conduct of the study in the present form.

Remarks:

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

There was no member of the study team who participated in voting / decision making process. The ethics committee is organized and operated according to the requirements of Good Clinical Practice and the requirements of the Indian Council of Medical Research (ICMR).

Sincerely,

Mala Ramanathan
Member Secretary, IEC

S No	Name	Hosp NO	DOS	Age	Sex	
1	MASHOOD MH	398275	31/7/15	60		0
2	SUDARSANAN	395335	30/7/15	53		0
3	DIVAKARAN \	397454	20/8/15	69		0
4	SUDHEER	399548	3/8/2015	45		0
5	SHAHJHAN	393689	26/5/15	61		0
6	VARGHESE DI	376743	7/2/2014	68		0
7	JAFAR	389267	2/2/2015	61		0
8	SOMAN	273424	24/4/15	55		0
9	MOHANAN NA	393699	25/06/15	59		0
10	MURALIDHAR	396485	23/07/15	55		0
11	CHANDRAN V	386534	23/04/15	50		0
12	JAGANNATHA	393486	6/4/2015	68		0
13	RAJESHWARI	387546	20/04/15	68		1
14	DHARMAJAN	230618	22/04/15	66		0
15	BABU S	391526	27/04/15	53		0
16	JAYAKUMAR K	383680	29/04/15	53		0
17	LAKSHMANAN	265437	20/06/15	55		0
18	REGHUNATHA	391107	16/06/15	57		0
19	ARIFA BEEVI	390148	10/6/2015	53		1
20	DEEPTHI KUM	394638	22/06/15	45		0
21	MADHAVAKUI	393792	15/05/15	56		0
22	PADMAKARAN	389770	18/05/15	68		0
23	THEYYUNNI P	391779	7/7/2015	60		0
24	SEETHA A	388648	27/05/15	62		1
25	RAJAN G	385635	9/7/2015	55		0
26	SAVITHRY MK	391810	30/06/15	61		1
27	SAINUDHEEN	391674	1/7/2015	50		1
28	HAMEED M	393239	19/05/15	62		0
29	JOHN AS	379051	28/05/15	67		0
30	RAMACHANDI	394549	23/06/15	51		0
31	SUDHI KUMA	294098	15/07/15	51		0
32	THAMPY G GE	393681	30/05/15	53		0
33	GEORGE G	391438	26/06/15	63		0
34	SIVANANDAN	387248	22/02/2016	69		0
35	TIMOTHY AL	382161	20/07/15	62		0
36	RAJAN K	391541	14/10/15	64		0
37	SUVARNAKUM	317887	13/10/15	52		0
38	ARUMUGAM S	395392	30/10/15	50		0
				58.1578947		
				6.89898667		

Weight	Height	Comorbities	DM	HTN	DLP	
80	166			1	1	0
68.5	172			1	1	1
66.5	156			0	0	0
63	168			0	1	1
61	166			0	1	0
71	170			0	1	1
68	167	CAS		1	1	0
70.5	166			1	1	1
61.5	167			0	1	0
65	167	POST PCI		0	0	0
76	166	REC ACS		0	1	1
71	163			1	1	0
63	148			1	1	1
66	165	POST PCI		1	1	0
62	159			0	1	1
64	165			0	1	0
57.5	153	POST PCI		0	1	0
68	166			1	0	0
61	150	POST PCI		0	1	0
78	170	POST THROM		0	1	0
77.5	168	REC NSTEMI		1	1	1
58	162	REC NSTEMI		0	0	0
64	167			0	1	1
68	154			0	1	1
75	172	REC ACS		0	1	0
48	148.5			0	0	0
60.8	164	REC NSTEMI		0	1	0
66	170			1	0	1
81	174			1	1	1
62	167			1	1	1
67	164	POST PCI		0	1	1
65	163			0	1	1
77	170			0	1	0
62	158.5			1	1	1
65	168			1	1	1
58	164	POST RFA		1	1	1
63	166	POST IWMI		1	1	1
58	153	REC AWMI		1	1	0

66.2315789	163.763158
7.08729755	6.67461593

TTE	Pre CPB TEE	E velocity	A velocity	E/A	EDT
EF-67, DD-1, NO RWMA		55.2	45.8	1.2	222
EF-66, NO RWMA		44.9	37.8	1.2	187
70, NO RWMA		89.5	54.7	1.6	201
EF=68%, NO RWMA		51.4	48	1.1	194
EF=65%,NO RWMA		51.8	42.3	1.2	190
EF=66%, NO RWMA		45.3	73.3	0.6	298
EF=66%, NO RWMA		52.5	45.3	1.2	197
EF-36, RWMA+		81.9	26.1	3.1	155
EF=68, NO RWMA		43.5	34.2	1.3	132
EF=52%, NO RWMA		42.7	55.2	0.8	268
EF=68%, RWMA+		55.6	47.2	1.2	177
EF=69%, NO RWMA		65.2	75.2	0.9	208
EF=59%, RWMA+		38.3	49.1	0.8	232
EF=56%, RWMA+		49.7	73	0.7	150
EF=69%, RWMA+		74.6	69	1.1	160
EF=64%, NO RWMA		40.6	31.5	1.3	140
EF=69%, NO RWMA		48	66.3	0.7	254
EF=68%, RWMA +		63.7	51.6	1.2	182
EF=63%, NO RWMA		55	38	1.5	211
EF=47%, RWMA+		49.1	71.7	0.7	245
EF=69%, NO RWMA		52.5	33.8	1.6	176
EF=66%, NO RWMA		64.6	49.7	1.3	232
EF=73%, NO RWMA, DD-1		50.7	47	1.1	195
EF=72%, NO RWMA		46.4	60.4	0.8	225
EF=53%, RWMA+		65.3	36.2	1.8	153
EF=72%, RWMA+		36.8	53.3	0.7	254
EF=56%, RWMA+		60.8	52.6	1.2	176
EF=52%, RWMA+		39.9	55.2	0.7	171
EF=58%, RWMA+		75	64	1.2	180
EF=52%, RWMA+		48.7	41.4	1.2	245
EF=68%, NO RWMA		46.4	38.7	1.2	169
EF=69%, NO RWMA		51.2	47.9	1.1	182
EF=68%, NO RWMA		69	59	1.2	137
EF=42%, RWMA+		56.4	54.1	1.04	169
EF=54%, RWMA+		40.2	43.6	0.9	176
EF=55%, NO RWMA		88.7	76.8	1.2	215
EF=65%, RWMA		46.5	34.6	1.3	151
EF=56%, RWMA		48.6	76.2	0.6	212
		54.9			
		13.4253089			

E'	E/E'	PV S/D	GD of DD	EF(2D)	EF(3D)
9.37	5.9	0.9	0	69	68.5
10.7	4.2	1.2	0	60	60.5
7.46	12	1.3	2	71	75.5
12	4.28	1.2	0	67	68.8
8.2	6.9	1.2	0	67	56.1
9.49	4.8	2.1	1	60	57.4
11.2	4.7	1.6	0	65	66.4
4.66	17.6	0.7	3	35	30
10.8	4	1.1	0	70	59.4
9.06	4.9	1.8	1	50	43.4
13.3	4.18	1.9	0	40	33
8.42	7.7	1.3	1	70	72
6.53	5.9	1.6	1	62	66
7.18	6.9	2	2	51	50
9.97	7.5	1.7	2	56	61.6
10.8	3.8	0.9	2	70	75.8
9.66	5	1.3	1	48	42.6
11	5.8	1.2	0	64	60
11.5	4.8	1.1	0	59	50
7.84	6.3	1.9	1	41	42.5
11.9	4.3	1.7	0	62	56
6.9	9.4	0.9	2	59	51.1
13.1	4	1	1	66	67.5
10.6	4.4	1.6	1	58	44.7
11.8	5.5	1.9	0	62	68.6
6.64	5.5	2	1	61	66.3
11.3	5.4	0.9	0	51	37.6
9.69	4.1	1.6	1	48	29.4
12	6.3	1.1	0	60	67
8.2	5.9	1.6	1	50	30.2
10.2	4.5	1.4	0	65	64.4
11.3	4.5	1.4	0	66	66.9
11.7	5.9	0.6	2	55	55.5
6.5	8.67	0.8	2	45	36.8
9.05	4.4	1.1	1	59	60
9.66	9.5	1.2	1	52	64.6
13.4	3.5	0.9	0	40	42.5
8.28	5.9	1.5	1	54	54
9.77263158				57.5789474	55.3315789
2.11530674				9.49717335	13.390499

CO	WMSI	RWMA	TMSV-16	TMSV-16 SD	TMSV-12
2	17	0	66	18	37
2.9	17	0	223	50	223
2.7	17	0	55	14	51
4	17	0	59	16	59
4.2	17	0	53	13	46
1.5	17	0	33	10	29
2.6	17	0	108	29	70
1.5	50	1	170	55	142
5.7	18	1	474	155	474
2.8	17	0	106	25	106
1.9	2	1	340	77	131
3.5	17	0	75	23	72
2.5	20	1	66	20	66
2.8	23	1	245	52	27
3.5	22	1	422	56	235
2.6	17	0	30	9	30
3.5	21	1	233	55	51
2.4	17	0	56	15	56
2	17	0	101	30	97
4.8	44	1	30	7	28
1.5	17	0	51	16	51
1.3	25	1	391	109	143
3.4	17	0	54	16	52
3.7	18	1	127	38	127
3.1	19	1	57	13	25
2.8	17	0	38	11	18
4	22	1	281	59	79
3.3	37	1	272	89	232
3.8	17	0	69	18	69
4.9	21	1	308	67	308
3.2	17	0	54	16	54
4.3	17	0	76	20	71
4.7	17	0	38	11	35
2.3	2.29	1	167	51	167
2.2	17	0	61	16	61
4.2	17	0	87	24	64
2.6	33	1	258	74	217
2.7	17	0	62	18	62

3.08947368	19.7707895
1.05081422	8.73119241

TMSV-12 SD	TMSV-6	TMSV-6 SD	RR-16	RR-16 SD	RR-12
11	29	10	5.72	1.57	3.18
57	204	72	22.68	5.18	22.68
13	45	17	6.25	1.59	5.78
17	28	11	6.6	1.75	6.6
12	25	8	5.14	1.29	4.43
10	29	11	4.03	1.25	3.56
23	67	27	11.89	3.21	7.72
48	142	54	22.15	7.09	18.45
177	466	230	47.38	15.49	47.38
28	106	34	9.33	2.19	9.33
51	120	57	40.34	10.41	39.53
22	33	14	7.48	2.33	7.23
22	66	29	7.93	2.43	7.93
8	17	6	30.63	6.52	3.32
56	235	79	50.64	9.59	28.26
10	30	11	3.25	0.97	3.25
15	51	20	24.47	5.74	5.32
15	56	22	4.68	1.22	4.68
31	97	39	9.77	2.92	9.37
7	28	10	6.45	1.55	6.11
16	47	18	5	1.57	5
47	109	43	32.56	8.6	11.89
15	36	16	5.27	1.57	5.06
44	113	50	15.68	4.71	15.68
8	12	5	5.5	1.3	2.45
5	17	6	4.59	1.36	2.13
21	79	27	44.98	9.39	12.65
79	193	85	27.61	9.03	23.62
19	38	13	7.29	1.86	7.29
77	241	105	31.27	6.85	31.27
18	54	24	6.27	1.86	6.27
22	71	30	8.84	2.38	8.29
12	31	14	3.79	1.13	3.47
54	114	42	15.05	4.61	15.05
17	61	20	5.39	1.41	5.39
18	40	15	9.58	2.63	7.04
50	98	37	30.11	8.59	25.28
18	38	15	9.95	2.89	9.95

RR-12 SD	RR-6	RR-6 SD	Exc-AVG	Exc-SD	Exc-MAX
0.95	2.51	0.84	7.1	3.1	14.9
5.77	20.74	7.31	5	2.4	12.2
1.51	5.05	1.9	8.2	3.3	15.5
1.88	3.09	1.27	6.3	2.1	10
1.12	2.45	0.79	5.1	4.1	14.9
1.2	3.56	1.34	4.8	3.6	12.8
2.49	7.4	2.98	4.8	4	13.8
6.19	18.45	7.08	2.8	2.6	10
17.71	46.57	23.03	4.2	2.8	10.4
2.46	9.33	3.02	3.2	3.4	9.4
10.32	38.35	9.85	2.9	1.8	5.8
2.23	3.34	1.42	8	5.6	23
2.68	7.93	3.52	4.8	2.3	11.5
0.98	2.08	0.76	4.2	2.2	8.2
6.75	28.26	9.42	6.3	4.2	15
1.07	3.25	1.22	7.9	3.2	15.2
1.53	5.32	2.13	4.3	2	9.3
1.24	4.66	1.81	4.5	2.8	10.3
3.02	9.37	3.78	3	2.6	7.6
1.58	6.11	2.2	4.4	2.4	9.4
1.6	4.65	1.79	5.5	2.9	13.8
3.96	9.11	3.55	3.4	3.1	11.4
1.49	3.51	1.57	6.7	3.4	14.4
5.38	13.93	6.16	3.5	2	8.5
0.79	1.17	0.44	6.3	2.5	11.6
0.67	2.01	0.75	5.1	2.4	10.4
3.35	12.65	4.29	3.8	2.4	11.4
8	19.65	8.64	2.1	1.4	5.1
2.02	4.01	1.39	6.4	2.9	12.9
7.83	24.53	10.71	2.5	3.3	9.8
2.1	6.27	2.76	5.5	2	9.1
2.55	8.29	3.55	7.4	4.1	15.4
1.18	3.09	1.35	5.4	2.3	9.5
4.87	10.28	3.77	2.8	2.1	7.4
1.53	5.39	1.8	4.8	2.8	10.5
1.95	4.41	1.67	4.7	1.9	8
5.89	11.45	4.27	3.4	3.6	10.7
2.89	6.15	2.41	4.1	2.3	10.1

Exc-MIN	PRE CPB RWM	POST CPB TEI	EF-1 (2D)	EF-1(3D)	CO-1
1.3	1		60	65.3	4.4
1.3	1		67	78	6.6
2.5	1		58	59	4.7
1	1		66	64.3	3.4
2.5	1		50	43.2	4
2.4	1		60	55	3.5
6.3	1(EAS)		62	68.3	3
2.5	1		30	32.1	3.2
2.5	0		53	63.2	5.9
3.7	1(EAS)		55	51.4	4.4
2.3	1		40	59.2	3
2.2	1		67	51	3.8
1	0 3D WRONG		65	63	3.1
2	1		50	50	4
2.7	1		62	71.1	4.5
0.4	1		74	63	4.3
0.6	1		32	50.5	3.2
2.1	1		54	50.4	2.6
3.6	0		64	67.5	3.1
1.5	0		50	57.9	4.2
1.5	1		55	48	3.4
2.3	1		41	44	3.5
3.3	1		54	58.4	5.3
2.2	0		73	52	5.8
0.4	0		49	56	10.4
1.3	1		61	63.1	5.7
1.9	1		52	32.3	4.8
2	1		48	35	4
0.1	1		51	47.4	4.2
6.5	1		58	47.2	5.7
0.8	1		70	58.7	3.1
1.2	1		64	70.2	3.7
1.5	1		65	72.8	5.2
2.2	0		50	52	3
2.3	1		59	55.7	3.9
1.7	1		63	60.6	4.3
3.4	1		49	51.9	4.7
0.9	1		51	41	3.1
			56.1052632	55.5184211	4.28157895
			10.0801906	10.903901	1.41189067

WMSI-1	RWMA	TMSV 16	TMSV-16 SD	TMSV-12	TMSV-12 SD
17	0	48	14	42	15
17	0	40	10	38	10
17	0	265	61	79	22
17	0	53	13	53	14
17	0	192	55	192	59
17	0	40	11	40	11
17	0	198	52	83	32
2.7	1	320	88	42	10
17	0	122	29	122	33
17	0	115	36	115	36
1.71	1	206	47	206	48
17	0	111	29	96	29
17	0	148	37	59	19
1.18	1	24	8	23	7
17	0	43	12	43	13
17	0	352	97	98	26
19	1	134	34	134	40
17	0	259	67	259	77
17	0	47	15	47	16
28	1	153	49	153	53
17	0	92	26	92	26
22	1	264	67	240	59
17	0	301	105	264	71
20	1	240	52	57	16
17	0	323	71	87	24
17	0	64	15	55	14
18	1	249	76	199	52
33	1	201	52	201	48
17	0	88	24	52	17
17	0	327	75	162	42
17	0	324	76	118	35
17	0	36	9	36	9
17	0	61	18	61	20
2.06	1	320	110	304	90
17	0	65	17	65	18
17	0	141	38	138	40
25	1	157	41	157	47
17	0	105	28	105	30

16.6223684
6.12114432

TMSV-6	TMSV-6 SD	RR-16	RR-16 SD	RR-12	RR-12 SD
40	19	7.06	2.11	6.24	2.25
17	7	4.84	1.23	4.55	1.17
63	27	32.69	7.56	9.73	2.75
53	20	7.49	1.79	7.49	2.04
136	49	25.96	7.37	25.96	8.03
40	14	4.86	1.32	4.86	1.32
46	10	33.63	7.15	11.18	3.08
42	10	40.12	10.5	14.2	2.88
122	44	18.12	4.29	18.12	4.84
26	9	14.19	4.44	14.19	4.49
91	33	38.45	8.69	38.45	8.9
86	37	14.28	3.7	12.33	3.76
29	11	16.8	4.2	6.68	2.1
19	7	3.56	1.1	3.4	0.98
8	3	6.22	1.76	6.22	1.93
35	13	42	11.62	11.7	3.12
134	64	20.1	5.09	20.1	5.94
259	100	52	13.5	52.3	15
29	10	5.73	1.81	5.73	1.91
46	18	24.27	7.69	24.27	8.35
26	9	13.23	0.83	13.23	3.73
129	49	34.29	8.77	31.17	7.7
47	22	37.1	12.96	32.61	8.75
24	8	36.8	7.99	8.8	2.44
87	30	47.85	10.5	12.97	3.55
34	12	8.89	1.3	7.57	1.88
78	31	36.51	11.17	29.18	7.66
101	36	33.53	8.64	33.53	7.94
35	14	12.96	3.5	7.62	2.47
82	28	55.6	12.74	27.56	7.12
66	23	43.16	10.14	15.17	4.62
30	11	4.98	1.17	4.98	1.27
61	27	7.16	2.1	7.16	2.33
72	31	40.01	12.1	38.52	10.12
32	10	8	2.05	8	2.23
62	26	19.09	5.09	18.59	5.46
129	52	23.76	6.15	23.76	7.15
64	25	13.61	3.7	13.61	3.89

RR-6	RR-6 SD	Exc-AVG	Exc-SD	Exc-MAX	Exc-MIN
5.97	2.83	5.1	2.4	9.5	2
2.09	0.79	5.8	3.6	11.9	2.4
7.79	3.28	4.2	4.5	15.4	3.8
7.49	2.88	6.3	3.8	14.4	3
18.41	6.67	3.8	4.3	11.2	8
4.85	1.64	4.7	2.5	9.8	1.7
1.68	0.62	4	3.5	11.4	2.4
8.9	2.1	2.8	2.6	8.2	2.6
18.12	6.54	4.4	3.4	11.7	3.4
3.26	1.13	5.2	4.4	16.7	3.8
17.07	6.16	5.7	4.7	17.1	3.5
11.61	4.75	5.2	4	17.3	3
3.33	1.28	3.4	2.7	9.2	2
2.72	0.98	4.3	3.6	11.2	4.6
1.2	0.5	6.9	1.8	11.5	0.9
4.17	1.56	3.8	3.8	10.5	4.2
20.1	9.64	3.6	4.6	13	7.6
52.31	20.26	5.2	4.1	14.3	3.2
3.47	1.27	3.8	2.6	9.7	3.2
7.33	2.88	5.1	3.1	10.1	1.8
3.66	1.3	3.8	2.4	9.8	1.6
16.77	6.42	3.9	5.4	13.8	7.6
5.81	2.67	5.3	5.7	18.1	8.6
3.7	1.3	4	1.7	7.8	2.1
12.97	4.52	4.2	2.8	9.5	3.1
4.77	1.65	4.5	1.8	7.6	0.8
11.51	4.52	3.1	2	7.8	2.2
16.86	6.07	3.1	2.1	7.4	2.6
5.15	2.11	3.1	1.8	7.6	1.2
13.87	4.84	2.5	3.4	9.5	3.2
8.79	3.03	4.7	3.7	12.7	5.7
4.1	1.54	7.9	3.8	18.9	0.3
7.16	3.15	8.2	3.3	15.1	1.3
8.98	4.92	3.4	2.7	10	2.2
3.89	1.29	4.9	4.7	14.8	2.7
8.35	3.55	5.3	3.5	12.9	1.7
19.53	7.84	5.3	4.6	14.7	4.4
8.34	3.3	3.4	2.4	8.6	2.9

POST CPB RW	POST ADR TE	EF-2 (2D)	EF-2(3D)	CO-2	WMSI-2
1		60	50	4.9	17
1		62	68.3	4.7	17
0		70	62.4	5.6	17
1	NIL	NIL	NIL	NIL	NIL
0		55	53.1	4.5	17
1		62	56.6	4	17
0		60	51	2.3	17
1		25	30	3	45
0		66	60.5	7.3	17
0		55	51	3.8	17
1		42	32	3	1.53
0		70	73	4.8	17
0		70	72	2.9	17
1		55	29.3	4.8	23
1		57	63	6	1
0		77	84	4.6	17
1		66	77.1	5.6	17
0	NIL	NIL	68.4	2.1	17
1		57	55	6	17
1		42	52.7	4.1	18
1		60	58.2	9.5	17
1		78	78	4.4	17
0		54	67.5	4.7	17
1		72	68.5	5.5	17
0		69	46	4.6	17
1		57	67.6	4.4	17
1		59	34.3	2.1	18
1		45	36.3	4.4	32
1		55	45.6	4.5	17
0		60	55.8	4.9	17
0		67	63.9	3.9	17
1		68	73.4	6	17
1		67	56.3	5.9	17
1		51	54.7	3.6	2.06
1		58	58	4.7	17
0	NIL	NIL	NIL	NIL	NIL
1		42	48.13	4.5	24
0		54	61	2.4	20
		59.0571429	57.2952778	4.55555556	17.4052778
		10.9220308	13.7711791	1.46802967	7.2063692

RWMA-2	DD-GD	E VEL	A VEL	E/A	EDT
	0	2	64.1	64.1	1	183
	0	0	84.5	52.5	1.6	158
	0	2	88.9	59.6	1.5	201
NIL		0	45.5	42.4	1.1	183
	0	0	71.3	59.6	1.2	182
	0	0	68.5	50.8	1.3	180
	0	1	70.1	64.1	1.1	165
	1	3	107	34.5	3.1	150
	0	0	66.6	57.3	1.2	103
	0	1	65.7	84.5	0.8	169
	1	1	57.2	63.1	0.9	260
	0	1	70.6	107	0.7	240
	0	1	34	34.5	1	146
	1	2	73	66.1	1.1	116
	1	2	108	90.6	1.2	162
	0	0	64.1	53.6	1.2	148
	0	1	63.6	73.5	0.9	235
	0	2	72.6	70.4	1.2	150
	0	0	79.5	53	1.5	180
	1	1	74	81.4	0.9	228
	0	0	79	76.3	1	187
	0	2	77.3	66.3	1.2	176
	0	1	43.1	54.8	0.8	254
	0	1	50	80.7	0.6	211
	0	0	91.1	83.5	1.1	162
	0	1	57.5	71.7	0.8	264
	1	0	63	88.4	0.7	211
	1	1	48.6	77.3	0.6	201
	0	0	80.4	71.8	1.1	151
	0	1	69.7	70.3	1	285
	0	0	92.8	56.3	1.6	158
	0	0	61.4	75.5	0.8	173
	0	1	107	113	0.9	236
	1	1	63.5	90.5	0.7	190
	0	0	91.7	67.9	1.4	162
NIL		1	82.3	75.1	1.1	218
	1	0	88.9	79.5	1.1	162
	1	0	51.4	43.1	1.2	181

	71.7763158
	17.5472864

PV S/D	E'	E/E'	TMSV-16	TMSV-16 SD	TMSV-12
1	5.55	11.5	142	31	142
1.2	12.8	6.6	49	11	34
0.7	8.46	9.5	45	10	15
1	11	4.1	NIL	NIL	NIL
1.3	8.89	8	194	43	43
1.2	10.6	4.1	39	10	20
1.2	10.4	6.7	38	14	36
0.4	9	11.6	301	82	37
1.7	14.5	4.6	33	10	33
1.8	12.8	5.1	79	25	79
2.2	7.77	7.4	77	23	60
2.3	8.82	8	103	23	77
1.3	9.49	3.6	67	18	67
1.8	5.6	12.9	208	54	192
1.2	11.6	9.3	64	18	51
1.5	11.6	5.5	38	12	36
1.4	14.9	3.2	16	4	12
1.5	7	10.4	89	21	81
1.1	10.1	7.9	166	50	166
1.2	9.72	7.6	40	9	40
1.8	11.4	7.7	23	6	23
1.8	7.25	9.5	69	16	64
1.3	12.1	3.6	69	18	69
1.8	9.94	4.6	39	12	36
1.6	13.8	6.6	140	27	70
1.7	7.77	7.4	28	8	23
1.4	9.39	6.7	281	87	269
1.2	8.97	5.4	204	52	68
1	14.3	5.6	168	38	164
1.8	10.6	6.6	81	23	59
1.2	15.5	6	70	19	70
1.4	12.8	4.8	42	11	42
1.6	15.3	7	244	63	54
1.5	7.8	6.1	342	103	305
0.9	11.9	7.7	21	6	18
1.3	5.62	14.6	NIL	NIL	NIL
1.2	11.7	7.6	104	33	104
1.2	9.59	5.4	33	10	33

10.4297368
2.69908341

TMSV-12 SD	TMSV-6	TMSV-6 SD	RR-16	RR-16 SD	RR-12
33	142	49	25.52	5.64	25.52
10	33	12	6.69	1.52	4.58
5	6	3	7.38	1.62	2.37
NIL	NIL	NIL	NIL	NIL	NIL
13	19	7	29.76	6.53	6.64
8	20	8	5.32	1.35	2.71
13	36	16	6.12	2.25	5.68
12	37	13	36.59	9.97	4.44
9	33	14	5.41	1.66	5.41
27	45	17	9.73	3.02	9.73
19	54	19	13.46	4.03	10.51
21	55	19	14.71	3.27	10.99
19	60	23	9.24	2.5	9.24
57	58	23	32.87	8.6	30
14	31	14	9.36	2.65	7.47
12	29	11	4.98	1.55	4.64
3	8	3	2.36	0.67	1.77
22	34	13	12.2	2.82	11.01
99	166	55	19.1	5.78	19.1
10	14	6	6.39	1.49	6.39
6	12	4	3.43	0.83	3.43
17	61	22	8.69	1.99	8.02
21	66	25	4.97	1.32	4.97
12	36	16	6.32	1.94	5.91
18	16	6	33.17	6.42	16.58
7	23	8	4.45	1.3	3.67
84	109	47	40.81	12.65	39
21	68	24	37.34	9.59	12.41
42	164	59	23.01	5.22	22.38
19	41	15	13.66	3.9	9.98
20	35	13	9.97	2.73	9.97
12	39	15	5.91	1.55	5.91
15	54	20	30.09	7.79	6.7
82	79	30	38.77	11.72	34.52
6	12	5	3.91	1.16	3.24
NIL	NIL	NIL	NIL	NIL	NIL
37	104	51	14.55	4.69	14.55
10	25	10	4.49	1.35	4.49

RR-12 SD	RR-6	RR-6 SD	Exc-AVG	Exc-SD	Exc-MAX
6.03	25.52	8.86	3.8	2.1	8.7
1.34	4.45	1.7	5.6	1.5	10.5
0.81	1.02	0.42	4.7	2.7	11.7
NIL	NIL	NIL	NIL	NIL	NIL
1.96	2.98	1.12	3.8	2.6	9.3
1.03	2.68	1.09	4.8	3.5	9.2
2.08	5.68	2.5	3.1	2.4	7.3
1.43	4.44	1.59	2.6	2.4	7.8
1.52	5.41	2.23	5.3	2.5	12
3.28	5.59	2.04	5.7	3	11.6
3.39	9.4	3.25	2.9	1.6	5.4
2.94	7.88	2.74	8.1	4.6	17.9
2.6	8.35	3.13	5.3	3.4	13.9
8.9	9.25	3.67	1.6	5.1	9.9
2.12	4.61	2.03	4.5	2.9	12
1.56	3.98	1.42	7.5	4.1	13.8
0.51	1.2	0.4	7.8	2.9	14.7
3.04	4.58	1.82	6.3	4	13.9
5.59	19.1	6.28	3.7	2.6	10
1.67	2.31	0.89	5.2	2	11.4
0.87	1.83	0.6	5.2	3.3	14.9
2.14	7.61	2.77	7.7	4.6	15.8
1.49	4.7	1.83	7.2	4.8	18.4
2.01	5.91	2.66	4	1.7	14.1
4.32	3.83	1.53	4	4.1	14.4
1.04	3.67	1.33	5.3	2.8	10.6
12.41	15.81	6.82	3.2	2.1	9
3.77	12.41	4.49	3	2.5	8.3
5.71	22.38	8.03	4	3.3	10.1
3.17	6.82	2.48	4.5	2.3	8.3
2.83	4.95	1.85	5.6	2.4	12.7
1.77	5.54	2.18	7.4	4.4	17.5
1.85	6.7	2.41	5.8	4.1	14.4
9.32	8.95	3.39	3.8	2.5	9.6
1.04	2.29	0.98	5.7	2.4	15
NIL	NIL	NIL	NIL	NIL	NIL
5.19	14.55	7.11	4.3	3.2	10.3
1.37	3.35	1.28	5.8	2.6	10.2

Exc-MIN		POST ADR RV	INTERVENTIC	IMPROVEMENT		
	1.9	0	1,ADR STOPP		1	ADR CAUSING IMBALANCE
	1.3	1	0		1	PRE CPB HIBERNATION IMP
	1.2	1	0		1	POST CPB STUNNING IMPR
NIL	NIL		ADR NOT STA	NIL		
	3	0	0		0	POST CPB STUNNING PERS
	4.9	1	0	NIL		DD IMPROVED, NO DYSSYN
	3.4	1	0		1	POST CPB STUNNING IMPR
	2.9	1	PDA & PLB NO		0	AKINETIC SEGMENTS NOT I
	0.9	1	0		1	PRE CPB HIBERNATION IMP
	2.6	1	0	NIL		EAS in post CPB-1, later SD
	2.8	1	0		1	SDI IMPROVED
	3.5	0	0		1	POST CPB STUNNING IMPR
	2	1	0		1	Pre CPB hibernation & POST
	10	1	ADDITIONAL		1	SDI IMPROVED WITH ADD (
	2.3	0 3D ERROR	ADR STOPPEI		1	PLB NON GRAFTABLE, ISCH
	2.1	1	0		1	POST CPB STUNNING IMPR
	1.8	1	0		1	PRE CPB HIBERNATION IMI
	3.2	NIL	ADR NOT STA	NIL		POST CPB EARLY ACTIVATI
	3.1	0	ADR STOPPEI	NIL		EARLY ACTIVATING AREAS
	1.5	1	0		1	PRE CPB HIBERNATION & P
	1.9	1	0	NIL		
	2.3	1	0		1	POST CPB RWMA + MR IMP
	2.6	1	0		1	POST CPB STUNNING IMPR
	0.7	1	0		1	POST CPB STUNNING IMPR
	4.6	1	0		0	POST CPB STUNNING IMPR
	3.1	1	0	NIL		
	2.5	1	0		0	NO CHANGE, NO WORSENI
	5	1	0		0	NO WORSENING
	6	0	0	NIL		
	1.4	1	0		1	PRE CPB HIBERNATION & P
	0.8	1	0	NIL		EAS IN POST CPB-1, LATER
	0.6	1	ADR STOPPEI	NIL		
	1.9	1	ADR STOPPEI	NIL		ADR CAUSING IMBALANCE
	2.2	1			0	INF WALL THINNED OUT
	1.4	1	ADR STOPPEI	NIL		
NIL	NIL		ADR NOT STA		1	EARLY ACTIVATING AREAS,
	2.5	1	0		1	PRE CPB HIBERNATION PAR
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Key to Master Sheet

1. S. No. – Serial Number
2. Hosp No. – Hospital Number
3. DOS – Date of surgery
4. DM – Diabetes Mellitus
5. HTN – Hypertension
6. DLP – Dyslipidemia
7. TTE – Transthoracic Echocardiography
8. Pre CPB – Pre cardiopulmonary bypass
9. TEE – Transesophageal Echocardiography
10. EDT – E wave deceleration time
11. PV S/D – Pulmonary vein S/D
12. Gd of DD – Grade of diastolic dysfunction
13. EF (2D) – Ejection fraction 2-dimensional
14. EF (3D) – Ejection fraction 3-dimensional
15. CO – cardiac output
16. WMSI – wall motion score index
17. RWMA – regional wall motion abnormality
18. TMSV-16 – Time to reach minimal systolic volume among 16 segments
19. TMSV 16-SD – Standard deviation of time to reach minimal systolic volume among 16 segments
20. TMSV 12 - Time to reach minimal systolic volume among 12 segments
21. TMSV 12-SD - Standard deviation of time to reach minimal systolic volume among 12 segments
22. TMSV-6 - Time to reach minimal systolic volume among 6 segments
23. TMSV 6-SD - Standard deviation of time to reach minimal systolic volume among 6 segments
24. RR 16 - Time to reach minimal systolic volume among 16 segments normalized to RR interval
25. RR 16-SD – Standard deviation of time to reach minimal systolic volume among 16 segments normalized to RR interval
26. Exc Avg – average value of excursion
27. Exc Max – maximum value of excursion
28. Exc Min – minimum value of excursion
29. Exc SD – standard deviation of excursion
30. EF-1 – Post CPB Ejection fraction without adrenaline
31. CO-1 – Post CPB cardiac output without adrenaline
32. EF-2 – Post adrenaline ejection fraction
33. CO-2 – Post adrenaline cardiac output
34. WMSI-2 – Post adrenaline WMSI

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