EFFECT OF SEVOFLURANE VERSUS PROPOFOL ON SEGMENTAL AND GLOBAL LONGITUDINAL STRAIN IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS SURGERY

Thesis submitted for the partial fulfillment for the requirement of the degree of

DM (Cardiothoracic and Vascular Anesthesiology)

of

S.C.T.I.M.S.T

DR. CHENNAKESHAVALLU G N

SENIOR RESIDENT

DM CARDIOTHORACIC AND VASCULAR ANESTHESIOLOGY

YEAR 2016-2018

DEPARTMENT OF ANESTHESIOLOGY,

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY,

TRIVANDRUM, KERALA, INDIA 695011

DECLARATION

I hereby declare that this thesis entitled, "**Effect of sevoflurane versus propofol on segmental and Global longitudinal strain in patients undergoing coronary artery bypass surgery**" has been prepared by me under the supervision and guidance of Prof. Shrinivas Gadhinglajkar, Prof. Rupa Sreedhar, Prof. Prasant Kumar Dash, Division of Cardiothoracic and Vascular Anesthesiology, Department of Anesthesiology, and Prof. Jaya Kumar. K, Professor and Head, Department of Cardiothoracic and Vascular Surgery, at Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram.

Dr.Chennakeshavallu G N

Senior resident

DM Cardiothoracic and Vascular Anesthesiology,

Department of Anesthesiology,

SCTIMST, Thiruvananthapuram.

Date:

Place: Thiruvananthapuram

CERTIFICATE

This is to certify that this thesis entitled, “Effect of sevoflurane versus propofol on Segmental and Global longitudinal strain in patients undergoing coronary artery bypass surgery” has been prepared by **Dr. Chennakeshavallu G N**, DM Cardiothoracic and Vascular Anesthesiology Resident, Division of Cardiothoracic and Vascular Anesthesiology, at Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram. He has shown keen interest in preparing this project.

(GUIDE)

PROF. SHRINIVAS V GADHINGLAJKAR

Professor in Anesthesiology, SCTIMST, Thiruvananthapuram

(COGUIDE)

PROF. RUPA SREEDHAR

Professor in Anesthesiology, SCTIMST, Thiruvananthapuram

(COGUIDE)

PROF. PRASANTA KUMAR DASH

Professor in Anesthesiology, SCTIMST, Thiruvananthapuram.

PROF. JAYAKUMAR K

Professor and Head, Department of Cardiothoracic and Vascular Surgery,

SCTIMST, Thiruvananthapuram.

CERTIFICATE

This is to certify that this thesis entitled, "**Effect of sevoflurane versus propofol on segmental and Global longitudinal strain in patients undergoing coronary artery bypass surgery**" has been prepared by Dr. Chennakeshavallu G N, DM Cardiothoracic and Vascular Anesthesiology Resident, Division of Cardiothoracic and Vascular Anesthesiology, at Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram. He has shown keen interest in preparing this project.

Prof. Rupa Sreedhar,

Professor and HOD,

Division of Cardiothoracic and Vascular Anesthesiology,

Department of Anesthesiology,

SCTIMST, Thiruvananthapuram**.**

Date

Place: Thiruvananthapuram

ACKNOWLEDGEMENT

It is my proud privilege to express my deep sense of gratitude to **Professor Shrinivas V Gadhinglajkar**, Professor in Anesthesiology, Division of Cardiothoracic and Vascular Anesthesiology, Department of Anaesthesiology, S.C.T.I.M.S.T, Thiruvananthapuram, without whose constant encouragement, active guidance, valuable advice and cherished blessings, this study would never have seen the light of the day.

I take the opportunity to extend my immeasurable gratitude to **Prof Rupa Sreedhar**, Professor in Anesthesiology and Head of department, Division of Cardiothoracic and Vascular Anesthesiology, Department of Anaesthesiology, SCTIMST, Thiruvananthapuram, who has always been a fountain of inspiration and an unfailing source of guidance.

I am highly obliged and grateful to **Prof.Prasanta kumar Dash**, Division of Cardiothoracic and Vascular Anesthesiology, Department of Anaesthesiology, SCTIMST, Thiruvananthapuram, who extended his unstinted support in the preparation of my thesis.

I sincerely record my indebtedness to **Dr. Saravana Babu** for his contribution of the basic fundamentals of the statistical methods applied in this study

I have much pleasure in expressing my grateful thanks to the faculty members of the Division of Cardiothoracic and Vascular Anesthesiology of SCTIMST, Thiruvananthapuram **Prof. Thomas Koshy, Dr.Suneel P.R., Dr. Unnikrishnan K.P, Dr. Subin Sukesan** for their constant inspiration and valuable suggestions.

Very little of what is worthwhile in this thesis could have been written without the generous co-operation, patience, advice and assistance of **Dr. Sruthi Sankar**, Senior resident, Department of Anaesthesiology,SCTIMST, Thiruvananthapuram.

CONTENTS

ABBREVIATIONS

LV	=	Left ventricle
CABG	=	Coronary artery bypass grafting
CAD	=	Coronary artery disease
TEE	=	Transesophageal echocardiography
EF	=	Ejection fraction
2D	=	2-dimensional
GLS	=	Global longitudinal strain
STE	=	Speckle tracking echocardiography
CAD	=	Coronary artery disease
ASE	=	American society of Echocardiography
WMSI	=	Wall motion severity index
PSLS	=	Peak systolic longitudinal strain
TDI	=	Tissue Doppler Imaging
RWMA	=	Regional wall motion abnormality
LVOT	=	left ventricular outflow tract
ROI	=	Region of interest
ME	=	mid-esophageal
CPB	=	cardio-pulmonary bypass

INTRODUCTION

INTRODUCTION

Left ventricular (LV) systolic function is a predictor for perioperative morbidity and mortality in patients undergoing coronary artery bypass grafting (CABG) [1-3]. Transesophageal echocardiography (TEE) is a corner-stone for assessing LV systolic function in routine intraoperative practice. A reproducible and consistent quantifiable measure of regional and global myocardial function might improve preoperative risk stratification and guides anesthetic management when changes in myocardial function occur. Conventional methods for analysis of regional and global LV systolic function use qualitative/quantitative approach like visual assessment of inward radial motion and wall thickening, fractional shortening , fractional area change , stroke volume , Ejection fraction (EF) from 2-dimensional (2D) or Doppler evaluation of cardiac structures. These methods have many limitations including necessity to make geometric assumptions, less reproducibility, inter-observer variability, load-dependence, influence of hemodynamic variables and translation motion[4, 5]. Echocardiographically derived wall motion severity index has good ability to predict outcomes after CABG[6]. However, this method has limitations which include need for significant expertise, lack of precise distinction between varying degrees of hypokinesia and potential failure to identify subtle abnormalities[7]. Endocardial wall motions reflect only the radial mechanics of the heart neglecting the contribution of longitudinal myocardial deformation which is affected at early stages of ischemia. Furthermore, impairment of regional wall motion score does not cause reduction in LVEF unless several segments are involved. Also, tethering of a normally contracting tissue to the adjacent infarcted area may also appear akinetic accounting for overestimation of infarct size by WMS[8]. Several studies suggest that LVEF is poorly sensitive in detecting early myocardial dysfunction[9].

There is a growing body of evidence showing that the assessment of myocardial deformation by Doppler or speckle tracking techniques is a sensitive and robust index to detect subclinical myocardial dysfunction with defined normal range and provides incremental information in the clinical setting[10]. Doppler-based myocardial velocity, strain, and strain rate analyses have been suggested to quantify regional myocardial function by studies based on transthoracic echocardiography[11]. However, important limitations of Doppler-derived parameters are the considerable angle dependency and substantial noise artifacts in the operating room.

The recent technologic advances in signal processing in ultrasound (2D Speckle tracking imaging) enable measurement of tissue velocity and deformation in one or two dimensions, which provide high-quality , precise, and objective information regarding regional and/ or global myocardial function in real time, decrease the subjectivity of the interpretation and increase the diagnostic accuracy. Global longitudinal strain (GLS) identifies subclinical LV dysfunction in a number of circumstances, including ischemic heart disease[12]. GLS using two-dimensional speckle tracking echocardiography (2D-STE) by obtaining quantitative information with less inter-operator variability has many advantages over traditional wall motion scoring as mentioned above. During the interpretation of Dobutamine stress echocardiography in patients with ischemic heart disease, GLS has been found more sensitive than traditional WMS [13, 14]. Since the LV subendocardial area is most vulnerable to the effects of hypoperfusion and ischemia, the GLS/ Segmental longitudinal strain may therefore be attenuated in early stages in coronary artery disease (CAD) patients[15].

During cardiac surgery, inhalational anesthetic preconditioning has been found to protect the myocardium from injury [16, 17]. Similar cardio-protective benefits were also found to be offered by intravenous anesthetic drugs such as propofol through its anti-oxidant

effect. Propofol has been shown to markedly decrease the size of myocardial infarction, to lower troponin release, and to decrease the rate of mortality after cardiac surgery[18-20]. However, which anesthetic agents offer more cardio-protection after cardiac surgery is controversial[21-23]. 2D echocardiography was used in previously published intraoperative studies investigating the effects of sevoflurane and propofol in CAD patients[24-26].

The WMS may have limited sensitivity in detecting subtle changes in the myocardial contractility produced by administration of sevoflurane or propofol. Hence, another echocardiographic parameter may be indicated to identify these subtle changes associated with ischemia. As the STE has been introduced recently in cardiac surgical practice, there is scarcity of published data on its intraoperative utility in evaluating the effects of anesthetic drugs. To the best of our knowledge, the effect of anesthetic agents on peak systolic longitudinal strain in patients undergoing CABG has not been evaluated.

Therefore, this study was conducted with the objective of assessing whether segmental longitudinal strain with its ability to identify subtle derangement in LV systolic function induced by anesthetic agents (sevoflurane versus propofol) can be a substitute for already established RWMA grading (WMS)

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The current methods for evaluating intraoperative LV systolic function and their limitations

One of the major roles of intraoperative echocardiography, in context of CABG is to monitor LV function for detection of acute ischemia manifesting as new regional wall motion abnormality. Given the poor prognosis associated with LV systolic dysfunction, early detection is warranted[27]. The quantification of LV function is important in clinical cardiology and has implications for a variety of treatment decisions. In routine practice, conventional methods are used for analysis of regional and global LV systolic function like visual assessment of inward radial motion and wall thickening, fractional shortening, fractional area change, stroke volume, LVEF from 2D or Doppler evaluation of cardiac structures. LVEF remains the simplest and most widely used parameter for assessment of LV function. LVEF is a strong predictor of mortality/prognosis in systolic heart failure and retains a central role in clinical decision making[28]. LVEF has limitations imposed by load-dependence and technical challenges related to accurate tracing of endocardial borders and necessity of making assumptions regarding LV geometry[29]. LVEF has been considered as being a relatively late marker of myocardial dysfunction[30]. Regional LV ventricular systolic function is traditionally based on visual estimation (eyeballing) and wall motion score as defined by ASE. However, such established parameters, which are regarded as the markers of regional contractility, have been found to be relatively insensitive for detection of subclinical myocardial dysfunction and are highly subjective to operator's experience or interpretation. Similarly many of the other proposed parameters for the assessment of LV systolic function like stroke volume, cardiac output are load-dependent and liable to variability in interpretation. Even though 3D

echocardiography (3DE) for LV function can overcome some of these limitations of 2D echocardiography, it too faces some of the constraints related to the technology. 3DE is not practiced widely, requires advanced echocardiography systems, generate relatively low-frame rates[31] and requires post-processing and highly trained personnel.

Necessity for accurate estimation of LV systolic function

CABG surgery is performed on > 600,000 patients annually throughout the world[32]. Intraoperative TEE plays a pivotal role in monitoring LV function in these patients. At present, the estimation of LV systolic function is relied upon traditional methods of 2D echocardiography such as LVEF, WMSI and Cardiac output. These methods fail to detect the LV systolic dysfunction at early stages. Studies [33, 34] in CAD patients has shown that WMSI were poorly associated in predicting major adverse cardiac events (MACE) and hospital admissions. GLS is more strongly associated with shorter time to MACE and hospital admissions signifying its incremental prognostic benefits against the conventional measures such as WMSI. Even LVEF has been reported to be less accurate than GLS in cardiovascular risk stratification. However, there is paucity in the literature depicting the utility of GLS in the intraoperative period to predict subclinical LV dysfunction.

Myocardial deformation and strain imaging

The complex left ventricular myocardial structure involves organization of the myocardial fibers in layers that forms a leftward helix in the subepicardium and rightward helix in the subendocardium. This fibre arrangement results in longitudinal (mitral annulus to apex), radial (epicardium to endocardium), and circumferential (tangentially to epicardium) cardiac motions during systole and diastole. This arrangement results in longitudinal lengthening, circumferential thinning and radial

thickening during systole, with opposite changes in diastole. When visualized from apex, the overall direction of LV rotation is clockwise at the base and counter clock-wise at the apex (Figure 1). This complex three-dimensional cardiac motion cannot be appreciated on conventional 2D-TEE imaging. The 2DE imaging display the function of only middle layer of myocardium in the form of inward endocardial excursion in ME views and thickening in TG views. During systole, significant amount of radial thickening (40%) and longitudinal shortening (14%) contribute to the effective myocardial contraction[35]. The subendocardial layer of myocardium which accounts for the longitudinal motion is the first one to get affected by the acute ischemic event[15]. These longitudinal motion (ME views) and circumferential motion (TG views) are difficult to ascertain on 2DE, although they are equally important. Most of these limitations can be overcome by assessing myocardial deformation using speckle tracking echocardiography.

Speckle tracking echocardiography (STE)

Speckle-tracking assesses myocardial movement and deformation by tracking the denoted "speckles" in echocardiographic images. A unique pattern of speckles also known as "fingerprint" bright and dark pixels in standard B-mode images consistently represent a small region in the myocardium. These speckles having characteristic constructive and destructive interference patterns, which are produced by the ultrasound signals reflected from inhomogeneous myocardial tissue can be tracked frame-by-frame throughout the cardiac cycle[36]. A software algorithm extracts displacement, velocity, SR, and strain within the defined myocardial segment. In tissue Doppler, the strain quantification remains dependent on the angle of insonation to the ultrasound beam whereas in STE the strain quantification is performed by measuring the displacement of the ROI relative to the preceding frames over a cardiac cycle. Hence, unlike tissue Doppler, the STE strain measurement is angle-independent. Speckle-tracking strain

calculates deformation in two axes and thus can measure strain simultaneously in the longitudinal and transverse direction from long-axis views, and radial and circumferential direction from short-axis views. Speckle-tracking strain is less affected by tethering or translation artifacts. Ischemic myocardial segments adjacent to the normally contracting segments may demonstrate displacement and velocity due to tethering effect. However, if deformation does not occur in the tethered segment, the regional strain and SR remain near zero[37]. This absence of strain and SR in the tethered segment distinguishes lack of active contraction from passive motion. Hence, STE strain provides robust measurements of myocardial deformation with acceptable intra-observer and inter-observer variability and may currently be regarded as reference method for the assessment of myocardial strain.

Limitations of STE strain quantification

STE is limited by its dependence upon the quality of echocardiographic images, which determines the ability to track the speckle pattern and endocardial borders. Inadequate tracking may occur because of complex 3D out-of-plane motion of the heart that makes it difficult to track speckles from image-to-image. Also, acoustic shadowing and reverberation artifacts interfere with frame-by-frame tracking, which decrease accuracy of measurement [38]. STE strain analysis depends on settings performed by the operator such as positioning the speckles at the ROI and defining the myocardial tracking, the quantification of global and regional strain is considered as a semi-automatic method. But despite of these limitations, speckle-tracking provides a simpler, more reproducible and angle-independent method of estimating strain in the operating room environment and serves as a good option for assessing the intraoperative regional myocardial function.

Quantification of the left ventricular segmental strain and GLS

ASE recommends[39] a 17-segment model of LV for assessment of regional LV function. Echocardiographic quantification of regional myocardial function is currently based on TDI or STE. The most commonly estimated deformation parameter is longitudinal strain during LV systole. The 17th segment (apical cap) is usually excluded from the assessment of WMS or regional strain. Specific normal values for regional LV strain are lacking at present. The reference values for 2D STE-derived regional deformation parameters are still under the domain of research.

Comparison of GLS with reference method for evaluation on LV systolic function

Cardiovascular magnetic resonance (CMR) is the gold standard for evaluation of cardiac volumes and function. Several studies have compared CMR and echocardiographic LV volumes and LVEF for assessing LV systolic function. Brown et al[40] showed a better correlation of GLS with LVEF measured on CMR than that of 2DE, and concluded that GLS is an effective method for quantifying global LV function, particularly in patients with extensive wall motion abnormalities. Aurich et al[41] in his study reported that quantification of LV strain on global and regional levels showed a better reproducibility and intermodal agreement with CMR.

Comparison of WMSI, LVEF with GLS

As stated above, the WMS reflects only the radial mechanics of LV contraction, ignoring the contribution of the longitudinal fibres. GLS takes into accounts the function of radial, transverse and longitudinal fibres that detects subtle changes in LV dysfunction at an earlier stage. Choi et al[42] demonstrated that PSLS has potential to predict the presence of left main or triple-vessel CAD in the absence of RWMA's on routine echocardiography. Kukucha et al[43] also had shown that strain analysis was more sensitive than WMS in distinguishing wall motion abnormalities between normally perfused and ischemia

segments. In patients with CAD, who were treated with revascularization procedure, Adamo et al[44] found that some of the patients had derangement in LVEF at 3-month follow-up despite improvement in LVEF at the time of discharge. Those patients who had improvement in LVEF and normal GLS at the time of discharge continued to have preserved LVEF at 3-months follow-up whereas those having abnormal GLS and improved LVEF at discharge had deterioration in LVEF at 3 months. Hence, the authors concluded that GLS have potential to predict derangement in LV function 3 months after the surgery.

Intraoperative literature on use of GLS

There is a scarcity in the literature describing intraoperative utility of STE for the evaluation of effects of anesthetic agents on LV function. The current literature on this issue is limited to the pre-clinical studies[45, 46].

Effects of Anesthetic agents on LV systolic function

The choice of the anesthesia technique does influence ventricular function during cardiac surgery. Several studies have suggested that the volatile agents-based anesthesia is more effective in reducing the mortality rate compared with total intravenous anesthesia in cardiac surgery[47, 48].Although, no randomized control trials (RCT) or meta-analysis have established the benefits of TIVA on improving the myocardial function in cardiac surgical patients, several meta-analysis [49, 50] performed in cardiac surgical patients and a large RCT performed in non-cardiac surgical patients[51] have failed to demonstrate any beneficial effects of volatile anesthetics on clinically relevant outcomes. Hence, the debate on which anesthesia technique is better in improving outcomes after cardiac surgery remains unsolved [52,53]. At present, the echocardiographic evaluation of effects of anesthetic agents on LV function is primarily performed using parameters such as WMS, cardiac index and LVEF.

Justification of our study

1. Cardiac anesthesiologists increasingly are involved in the role of intraoperative echocardiography during cardiac surgery. The evaluation of intraoperative cardiac function remains subjective to a large extent at present. In order to reduce the operator-dependent errors, automated techniques of regional and global functional evaluation such as strain quantification are desirable.

2. The effects of various anesthetic agents on segmental and global longitudinal strain have not been evaluated adequately, with exception of the study published by Jakobsen et al [54] which depicts the effect of ketamine induction on GLS in 11 patients undergoing CABG. There were significant decreases in GLS after induction (from 11.2% ±2.3% to 8.3% ± 2.6%, p = 0.005) suggesting reduction in global LV systolic function. Therefore, we planned to conduct a prospective study to evaluate the effects of sevoflurane and propofol on global and segmental longitudinal strain.

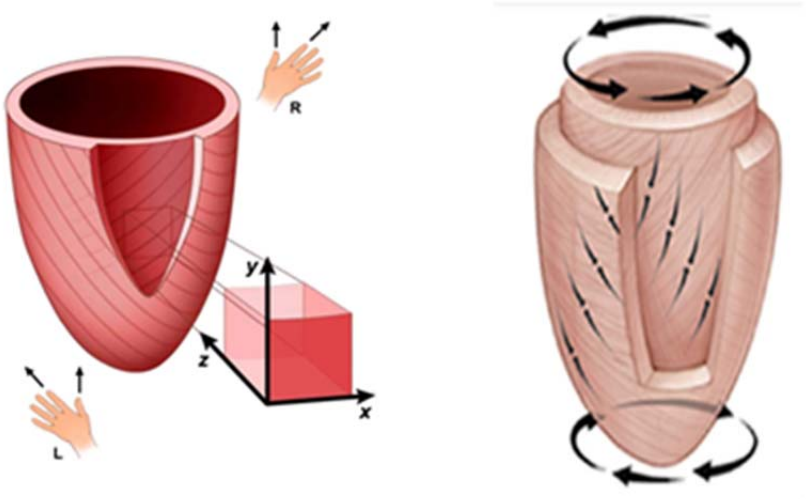


Figure 1- Myocardial architecture and deformation of the left ventricle. Arrangement of subepicardial layers in a leftward helix and subendocardial layers in a rightward helix leads to complex motion of the heart wherein there is clockwise rotation of the base and counterclockwise rotation of the apex of left ventricle

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

WMSI is the established method of evaluating the RWMA's in the intraoperative period, although it has many limitations. The STE is a new method useful for the same purpose, which may overcome the limitations of WMSI.

HYPOTHESIS

In this study we hypothesized that in patients undergoing CABG, the anesthetic agents (Sevoflurane and Propofol) reduce the peak systolic longitudinal strain (global/segmental) although regional wall motion remains unaltered.

Primary objectives

1. Using GLS/ Segmental strain in patients undergoing CABG, to quantify the effects of sevoflurane and propofol on LV systolic function

2. To demonstrate correlation between wall motion score index and global longitudinal strain

3. To evaluate the utility of GLS for predicting post-operative inotropic requirements (inotropic score)

Secondary objectives

1. To compare the effects of sevoflurane and propofol on conventional 2D echocardiographic parameters of LV systolic function (Wall motion score, Ejection fraction by Simpson's method, LVOT Cardiac output)

MATERIALS AND METHODS

MATERIALS AND METHODS

This prospective randomized study was conducted in a tertiary referral center, a University-level hospital annually operating about 1500 adult cardiac- thoracic patients. The study was approved by the Institutional ethics committee (IEC Regn No. ECR/189/Inst/KL/2013).

Patient selection

Patients scheduled to undergo elective, isolated, CABG surgery with cardio-pulmonary bypass were prospectively included in this study.

Inclusion criteria-

1. Adult patients undergoing isolated elective multiple graft (>2 grafts) CABG with cardio pulmonary bypass

2. Patients with good LV function (LVEF >55%)

3. Patients with sinus rhythm

4. Patients with right dominant coronary circulation as assessed by coronary angiography

Exclusion criteria

1. Emergency surgeries

2. Redo-surgeries

3. Patients with LMCA lesion >50%

4. Patients not willing to participate in study

5. Patients with recent acute coronary syndromes (<28 days)

6. Patient with moderate/severe LV dysfunction (LVEF<50%) and cardiac failure

7. Patient with moderate/severe mitral regurgitation or mitral stenosis

8. Patients with moderate/severe aortic regurgitation or aortic stenosis

9. Patients receiving pre-operative inotropes

10. Patients on pre-operative IABP and on mechanical ventilation

11. Patients requiring re-exploration due to surgical bleeding

12. Contraindication to TEE probe placement like esophageal strictures, esophageal varices, esophageal tumours, gastric ulcer, previous esophagectomy, esophageal diverticulum, tracheoesophageal fistula, previous bariatric surgery, hiatus hernia, large descending thoracic aortic aneurysm, unilateral vocal cord paralysis, esophageal varices, post‑radiation therapy

Randomization-

Randomization with numbered, sealed, opaque envelope to assign patients to one of the following 2 groups: propofol-based TIVA group (P -group) and sevoflurane group (S-group).

Informed Consent

Informed consent was obtained from all patients regarding the conduct of study. All patients were thoroughly examined during pre-anesthetic evaluation and educated regarding study protocol.

Anesthesia protocol

Patients were advised to continue all medications except oral hypoglycemic drugs, clopidrogel and ACE inhibitors. Oral diazepam 5-10 mg was administered for sedation the night before surgery. Patients in propofol group (P-Group) received TIVA throughout the surgery. Propofol (1-2 mg/kg, fentanyl (5-10 ug/kg), midazolam (0.1mg/kg) and pancuronium (0.1 mg /kg) were used for anesthesia induction. General anesthesia was maintained with propofol infusion (75-100µg/kg/min) throughout the procedure including CPB. Intermittent fentanyl boluses (to a total dose of 20 µg/kg/min) were administered to avoid changes of mean arterial pressure (MAP) within 20% from baseline but not less than 65mmHg. Bispectral index (BIS) values were maintained in the range of 40- 60. No volatile anesthetic was administered at any time during the procedure. The induction of general anesthesia in the sevoflurane group (S-Group) consisted of fentanyl (5-10 ug/kg), midazolam (0.1mg/kg), pancuronium (0.1mg/kg) and sleep dose of propofol (1-2mg/kg). Anesthesia was maintained with sevoflurane at a minimum end-tidal concentration of at least 1 minimal alveolar concentration throughout the entire procedure, including CPB, and was guided using BIS values between the ranges of 40-60. During CPB, sevoflurane was administered in the oxygenator circuit through a calibrated vaporizer. Fentanyl was administered to avoid changes of MAP within 20% from baseline but not less than 65mmHg. In pre- CPB period, any hypertension or hypotension were treated with nitroglycerin infusion or phenylephrine boluses to a target MAP of > 65mmHg. Patients requiring repeated boluses of phenylephrine were treated with nor-adrenaline infusion (0.05- 0.1 ug/kg/min). All patients were mechanically ventilated with volume control ventilation mode. Intraoperative continuous monitoring in both groups included 5-lead electrocardiography with ST analysis, heart rate, invasive blood pressure, pulse oximetry, body temperature,

respiratory gas monitoring, central venous pressure and BIS. All patients underwent CABG with CPB using JostraHL20 (Maquet, Rastatt, Germany) heart lung machines with a roller pump for non-pulsatile perfusion and membrane oxygenators with a flow appropriate for the core temperature. Electromechanical quiescence was achieved using intermittent antegrade cold blood cardioplegia. Two surgeons were involved in the study period as first operators. At CPB weaning, if MAP< 70 mm Hg, TEE guide LVOT CI < 2L/min/m^2, adrenaline infusion was commenced at a starting dose of 0.05 μg/kg/min. If desired targets not achieved, norepinephrine was added (starting dose of 0.05μg/kg/min). At the end of the surgical procedure, patients were transferred to the intensive care unit, where they were kept sedated with a continuous infusion of 20μg/kg/hr of morphine. When hemodynamically stable and rewarmed, the patients were weaned from the ventilator and extubated.

2D Echocardiography (2DE) - protocols

Pre-CPB: All studies were performed using the iE33 workstation (Philips Medical Systems, Andover, MA, USA) and RT-3D-TEE probes. Routine 2D examination was performed and the echocardiographic images were acquired at stable hemodynamic function (±20% of the basal values for the MAP and HR). LV volumes, Ejection fraction, stroke volume, cardiac output were assessed by 2D echocardiography, as recommended by the American Society of Echocardiography (ASE) [55]. Images were acquired from mid-esophageal (ME) 4-chamber view, 2-chamber view and long-axis view for RWMA and global/ segmental strain quantification with optimal gain and frame rate >50/s.

Post-CPB: All the examination mentioned above were repeated in post-CPB period at the time of skin closure.

Analysis of 2D echocardiographic parameters:

Quantification of LV volumes, Ejection fraction, Stroke volume, Cardiac output was performed on echocardiography system monitor in the operation theatre. WMSI was recorded in 3 ME views (4 C, 2 C, LV LAX view) and 3 TG views (basal SAX, mid papillary and apical view) using a 17-segment AHA model. For each segment, endocardial wall motion/ myocardial thickening was scored according to ASE guidelines-

Normokinesia = 1

Mild hypokinesia = 2

Severe hypokinesia = 3

Akinesia = 4

Dyskinesia = 5

At least 50% of the endocardial and epicardial borders or 100% endocardial borders were required to be visible in a segment for adequate analysis of the wall motion [56]. Global WMSI was calculated as follows-

WMSI= Σ score of 17 segments /17

WMSI was calculated separately for normal and ischemic segments based on coronary angiography report. WMSI was performed independently by two blinded experienced echocardiographers. An adjudicated consensus of WMSI was used based on joint review where there were discrepancies. In this study, WMSI of > 1.13 were classified as abnormal[57].

Longitudinal strain analysis:

Peak GLS and Segmental strain were estimated using video clips recorded at ME 4C view, ME 2C view and ME 3C (LAX) view with similar heart rate (within 10 beats/ min) as described by EACVI/ASE [39, 55]. A single lead electrocardiogram (lead I/II) was recorded simultaneously. Imaging depth and sector width were adjusted to achieve a frame rate > 50 frames/second. Images that presented the clearest visualization of the endocardium and epicardium border with the fewest artifacts were selected. Three cardiac cycles were analyzed for each imaging plane and stored as cine-loops in dicom format. For each measured or calculated variable, the averages of the three measurements were reported. Strain analysis was performed offline using a dedicated software package (QLAB 9, Philips Medical Systems - Andover, MA, USA) with the automatic delineation of endocardial and epicardial borders, tracking the speckles throughout the cardiac cycle (cardiac motion quantification), thus deriving the global longitudinal strain. A three-click method whereby the operator anchors three points, at each sides of the mitral annulus and at the apex of the LV for ME 4C view and ME 2C view, simplifies the process of tracking and analyzing peak systolic strain. For ME LAX view the points were placed at posterior mitral annulus, aortic annulus near RCC and LV apex. A color-coded parametric image that provides quick, visual impression of the extent of segmental myocardial deformation was generated. The timing of aortic valve closure was automatically determined. The quality of the tracking was visually assessed by the operator during motion playback. If necessary, the width of the region of interest was adjusted to the wall thickness excluding pericardium. Segments with inadequate tracking were rejected. The computerized assessment presented the data in bull's eye displays (figure 2). The segmental strain of ischemic / non- ischemic segments (based on coronary angiography), average GLS were noted separately using the software (figure 3).

All references to strain changes consider the absolute value of the number as percentage, so that higher or increase in longitudinal strain means a more negative number and lower or decrease means a less negative number.

The normal values for longitudinal strain were quoted from publication of Menting et al[33]. Different normal values were as follows, global peak systolic longitudinal strain (PSLS) of -17%, basal PSLS of -16 %, mid-PSLS of -18%, apical PSLS of -21% were considered as normal.

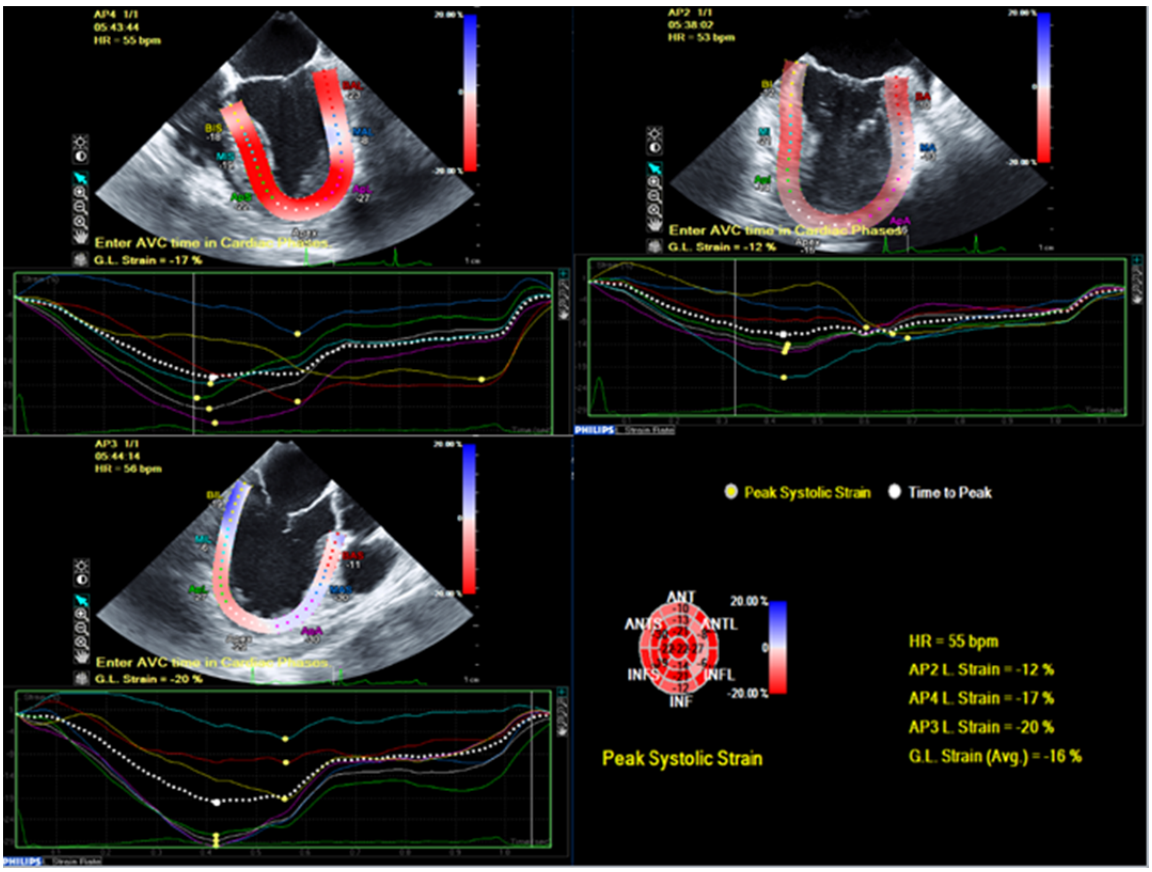


Figure 2 – Figure depicts an example of left ventricular peak systolic longitudinal strain measurement in a patient with triple vessel CAD. The left ventricle was traced in ME 4C, 2C & 3C views at end-diastole using ECG. The walls were automatically divided into seven segments at each view and global longitudinal strain at each view was calculated (A-C). Segmental PSLS was plotted in bull's eye and the LV GLS based on 17-segment model was calculated. Abbreviations – CAD- Coronary artery disease, ME- mid-esophageal, PSLS- peak systolic longitudinal strain, GLS- Global longitudinal strain.

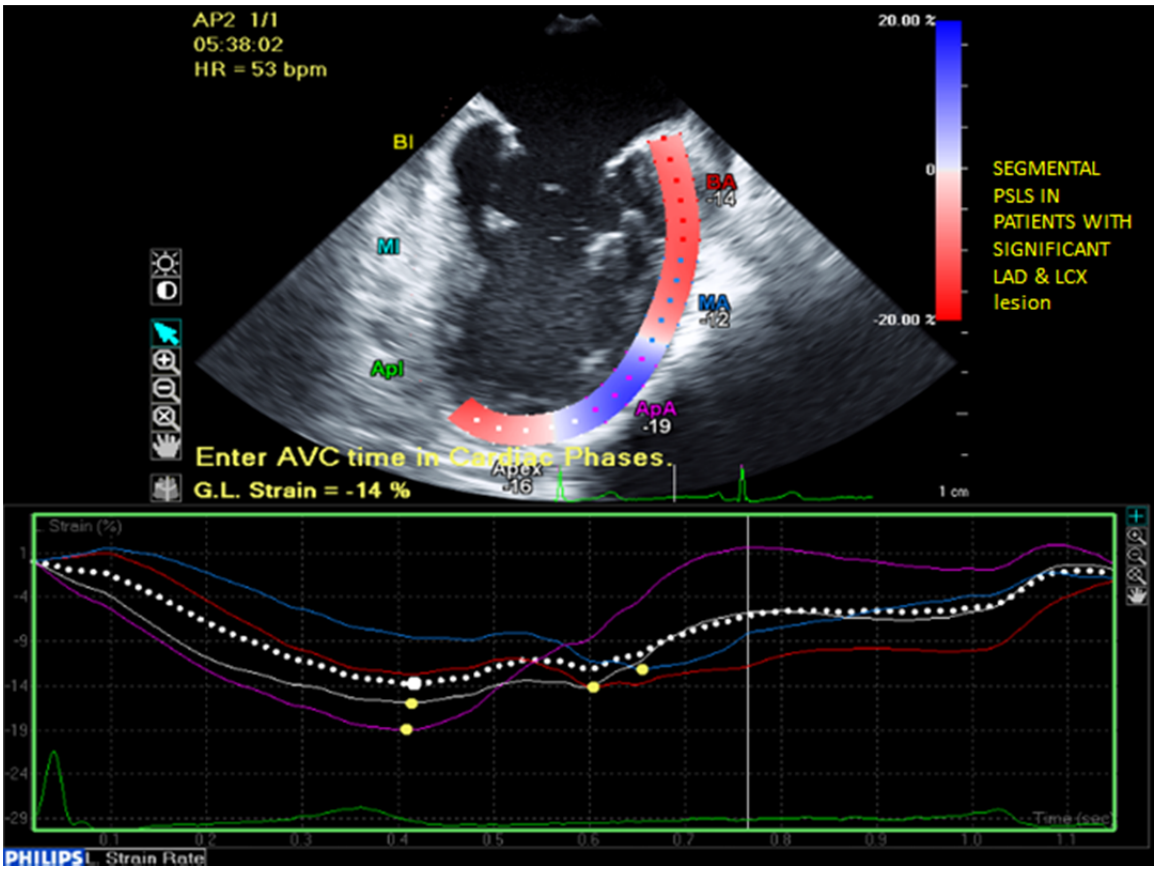


Figure 3 - Example of LV peak systolic longitudinal strain in a patient with double vessel disease (LAD & LCX involvement) demonstrating segmental PSLS values in ischemic regions only.

The study data was manually entered in the study chart as documented in annexures.

Preoperative Coronary angiography

Angiographic findings were assessed by an experienced interventional cardiologist who was unaware of patient's clinical and echocardiographic results. Coronary angiograms were visually assessed for all coronary lesions in two orthogonal planes. Lesion locations were assessed and percent diameter stenosis was measured for each coronary lesion according to the American Heart Association classification. In this study, angiographically-determined coronary artery disease was defined as percent diameter stenosis >70% for three epicardial vessels and >50% for LM coronary artery[43]. In our

study, the myocardial segments supplied by angiographically determined CAD were regarded as ischemic segments.

Postoperative parameters

Postoperative inotropic score[58] was calculated as follows

Inotropic score = 100 x adrenaline dose (μg/kg/min)+ 100 x noradrenaline dose (μg/kg/min).

Inotropic score exceeding 5.5 was referred as "high" and score below was referred as a "low"[59].

Duration of artificial ventilation and duration of ICU stay were noted.

STATISTICAL ANALYSIS

STATISTICAL ANALYSIS

The results obtained from the study were expressed in tabular format and graphs. Sample size was calculated based on a previous study "Effects of propofol, desflurane and sevoflurane on recovery of myocardial function after coronary surgery in elderly high – risk patients" [60]. For a power of 0.8 and α-error of 0.05, a sample size of 23 patients in each group was calculated to be appropriate. Quantitative variables such as Segmental strain, GLS, WMSI, LVEF, SVI, CI, inotropic score were expressed as mean, SD or median and inter quartile range. Qualitative variable (number of normal segments/ischemic segments) were presented as frequency and percentage. Between groups differences of quantitative variables were evaluated by unpaired t-tests. Inter-group comparisons of qualitative variables were analyzed by Chi square test. Correlations between quantitative variables were analyzed by Pearson correlation or Spearman Rank correlation. Pre-CPB and post-CPB comparison of quantitative variables were analyzed by paired t test or Wilcoxon sign rank test. A p value < 0.05 considered as statistical significant. Statistical analysis was performed using SPSS version 22.0.

RESULTS

RESULTS

In this prospective study, a total of 71 patients were included. Twenty-four patients were excluded for the following reasons: non-analyzable images in post bypass period (n= 16), frequent ventricular premature complexes / irregular cardiac cycle(atrial fibrillation) that precluded strain imaging in post bypass period (n=4). One patient required additional graft placement under second run of CPB, who also was excluded from the study.

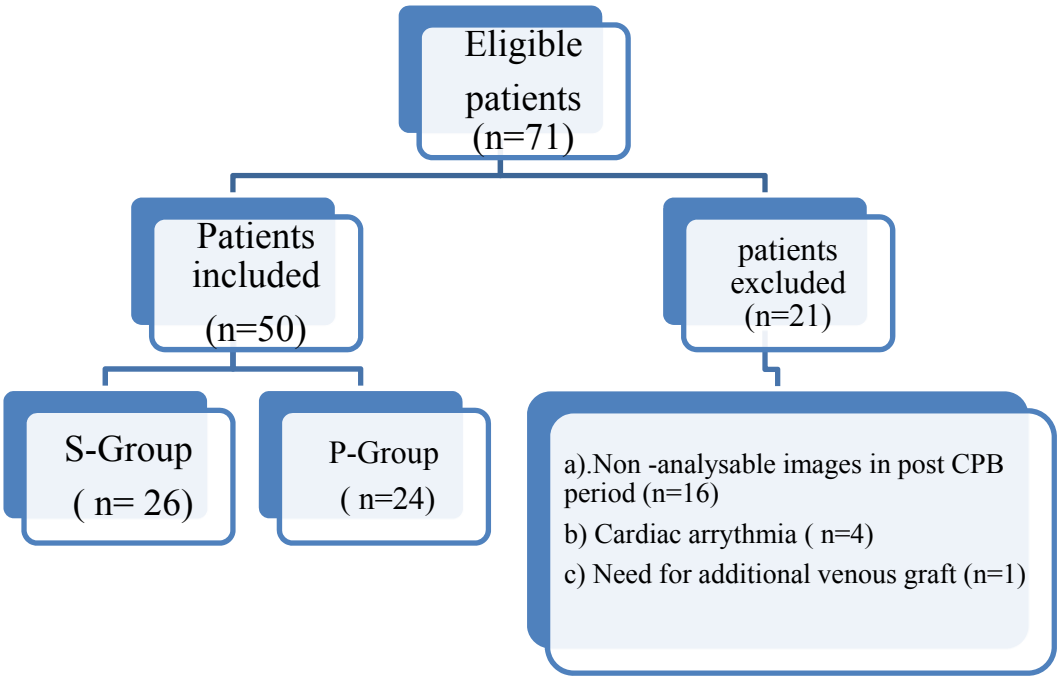


Figure 4- Consort diagram of patient selection

Demographic and clinical characteristics (Table 1) of the subjects (n=50) revealed that there were no differences in demographic details, co-morbidities and preoperative LVEF between the groups. In both the groups there was significantly higher percentage of subjects who were males. The percentage of normal and hypoperfused segments defined angiographically were equally distributed in both the groups. Intraoperatively, the number venous grafts, arterial grafts placed were statistically similar between the groups. Compared to sevoflurane group significantly more patients in the propofol group required nitroglycerin to control blood pressure in pre CPB period. The mean aortic cross clamp and CPB time was similar.

Hemodynamic details during the study period are displayed in Table 2. There were no significant inter group differences with regard to baseline heart rate and MAP. Average heart rate and MAP were similar in both the groups during pre-CPB and post-CPB period except the MAP which was significantly higher during pre-CPB period in sevoflurane. None of the patient presented tachycardia (HR.>100bpm) or ischemic changes through the study. The incidence of transient hypotension (SBP<90 mmHg) or bradycardia (HR<45bpm) were similar in both the group. The number of patients requiring nitroglycerine to achieve target MAP (around 70mmHg) were higher in propofol group. Compared to baseline, heart rate increased in post CPB period in both the groups but statistically insignificant (Table 3).

Table 1- Summary of demographic details, co-morbidities, coronary angiogram and preoperative and intraoperative characteristics in both groups

Parameter	S-Group (n=26) Mean±SD n(%)	P-Group (n=24) Mean±SD n(%)	P value (P<0.05 significant)
Pre-operative data			
Age , yrs	59.27±9.84	61.46±9.01	0.18
Sex , M/F	7/19(14%/38%)	2/22(4%/44%)	0.72
Body mass index(kg/m2)	22.4±4.2	21.3±4.3	0.15
Pre-op TTE Ejection fraction%	61. 61%±5.70%	59.87%±7.32%	0.85
Diabetes	20(40%)	18(36%)	
Hypertension	14(28%)	19(38%)	
Coronary angiogram			
No. of normal segments (lesion diameter <70%)	50 (5.88%)	61(7.17%)	1.94
No. of hypoperfused segments (lesion diameter >70%)	375(44.11%)	364(42.88%)	1.34
Intraoperative data			
NTG use prior to CPB	2	6	**<0.05***
No of venous bypass grafts (median)	4.15	4.13	0.215
No. of arterial grafts(median)	1	1	1
Aortic cross clamp, min	41.46±7.399	38.96±6.49	0.147
CPB time, min	106.92±10.1	106.83±15.49	0.15
Inotropic score (post CPB)	7.50±4.02	8.54±3.529	0.561

Table 1 shows demographic and clinical characteristics of the subjects (n=50). There were no differences in demographic details, co-morbidities and preoperative LVEF between the groups. Compared to sevoflurane group significantly more patients in the propofol group required nitroglycerin to control blood pressure in pre CPB period. The mean aortic cross clamp and CPB time was similar. (*) denotes p value <0.05 which was considered statistically significant.

Table 2- Hemodynamic comparison

Parameter	S-Group (n=26)	P-Group (n=24)	P value
Heart rate (bpm)			
Baseline	62.38±9.48	66.88±9.91	0.115
Pre CPB (average)	67. 69±10.45	72.25±10.21	0.126
Post CPB (average)	78.23±8.08	81.75±8.99	0.152
Mean arterial pressure(mmHg)			
Baseline	86. 62±11.76	89.83±11.11	0.326
Pre CPB (average)	74.73±8.34	68.54±8.52	**0.013***
Post CPB (average)	86.54±9.22	82.54±10.54	0.154

Table 2 shows hemodynamic parameters during the study period. There were no significant inter group differences with regard to baseline heart rate and MAP. The MAP was significantly higher during pre-CPB period in sevoflurane group. The number of patients requiring nitroglycerine to achieve target MAP (around 65 mmHg) were higher in propofol group. Compared to baseline heart rate increased significant in post CPB period in both the groups.

Table 3- Hemodynamic comparison within the group

	Baseline	Post-CPB	P value
	Heart rate(bpm)		
S- Group	62.38±9.48	78.23±8.08	0.091
P-Group	66.88±9.91	81.75±8.99	0.192
	MAP(mmHg)		
S-Group	86. 62±11.76	86.54±9.22	0.342
P-Group	89.83±11.11	82.54±10.54	0.247

Table 3 shows hemodynamic parameters within the group. There were no significant inter group differences with regard to baseline heart rate and MAP.

Effect of anesthetics on Conventional echocardiographic parameters

On comparing the preoperative and postoperative TEE data in both the groups, the LVEF and WMSI were found similar in both groups in pre and post CPB period. Calculated variables like SVI, CI also were comparable between the groups in pre and post CPB period (Table-4). Within the groups there was slight increase in LVEF in post CPB period which is statistically insignificant. There was no significant change in WMSI in both pre and post CPB period. In post CPB period, Cardiac index and SVI increased significantly in both groups ($p<0.005$) but increase was similar between the groups.

Table 4- 2D echocardiographic data between the groups

Parameter	**S-Group (n=26)**	**P-Group (n=24)**	P value
Pre-CPB			
Ejection fraction%	61. 61±5.70	59.87±7.33	0.231
WMSI	1.0685±0.145	1.061±0.094	0.543
Stroke volume index(ml/m^2)	43.96±11.07	42. 67±9.106	0. 655
Cardiac index (L/m^2/min)	2.93 ±0.599	3.129 ± 0.82	0.332
Post –CPB			
Ejection fraction %	65.07 ±8.01	64.41±7.54	0.284
WMSI	1.066±0.203	1.057±0.122	0.532
Stroke volume index(ml/m^2)	54.04±12.38	50.21±11.74	0.529
Cardiac index (L/m^2/min)	4.37±1.14	3.992±1.01	0.216

Table 4 describes the preoperative and postoperative TEE data in both the groups. There were no differences in LVLVEF, WMSI, SVI and CI between the groups in pre-CPB period. Similarly, no differences were found among these parameters between the groups in post-CPB period.

.

Table 5- Intra-group difference in 2D echocardiographic data

parameter	Pre-CPB	Post-CPB	P value
	Ejection fraction		
S- group	61. 61±5.70	65.07 ±8.01	0.543
P-group	59.87±7.33	64.41±7.54	0.084
	WMSI		
S- group	1.0685±0.145	1.066±0.203	0.742
P-group	1.061±0.094	1.057±0.122	0.061
	Stroke volume index (ml/m^2)		
S- group	43.96±11.07	54.04±12.38	0.002
P-group	42. 67±9.106	50.21±11.74	0.011
	Cardiac index(l/m^2/min)		
S- group	2.93 ±0.599	4.37±1.14	0.012
P-group	3.129 ± 0.82	3.992±1.01	0.031

Table 5- Pre- CPB and Post-CPB intra-group variables were compared using paired t-test. The results show no statistically significant differences within the groups.

Within the groups (Table 5) there was slight increase in LVEF in post CPB period which is statistically insignificant. There was no significant change in WMSI in both pre and post CPB period. In post CPB period, Cardiac index and SVI increased significantly in both groups ($p<0.005$) but increase was similar between the groups**.** On comparison of pre-CPB values for the range , mean and median segmental strain in both the groups, a wide variations in segmental PSLS were observed at all regions. In both the groups, average PSLS values were less negative in basal and mid segments than apical segments which demonstrated more negative strain. There was no statistical difference found between the groups in pre CPB period (Table 6). GLS values in sevoflurane group ranged from -11% to -24% with median value of -14%. In propofol group, median GLS was -13% with a range from -9% to -23%. There were no statistical significant differences between the groups. Similar results were observed in post-CPB period (Table 7). In postoperative period, GLS value range from -9% to -15% in sevoflurane group

with median value of -15% whereas the GLS ranged from -13% to -24% with a median value of -14%.

Table 6 **- Comparison of Pre-CPB strain values (Range, Median and Mean) between sevoflurane and propofol group in the cohort of patients**

	S- Group (n=26)			P-Group (n=24)			P value
	Range (%)	Median (%)	Mean±SD (%)	Range (%)	Median (%)	Mean±SD (%)	
BA	-6 to -19	-10	-15.23±3.52	-4 to -12	-9	-13.13±3.52	0. 616
BAS	-6 to -14	-14	-13.58±4.59	-5 to -18	-12	-12.44±4.59	0.571
BIS	-14 to -17	-15	-17.88±5.81	-7 to -20	-11	-16.76±5.61	0.855
BI	-7 to -18	-14	-15.13±8.48	-6 to -15	-12	-15.1±8.48	0. 603
BIL	-7 to -20	-13	-15±2.80	-7 to -22	-10	-13±2.80	0.786
BAL	-13 to -18	-16	-12.57±4.41	-13 to -21	-16	-11.87±4.41	0.877
MA	-12 to -20	-15	-14.5±4.55	-6 to -18	-14	-12.5±4.55	0. 608
MAS	-10 to -28	-15	-11.45±4.59	-5 to -20	-14	-12.75±4.59	0.089
MIS	-13 to -19	-17	-11.76±3.25	-13 to -21	-18	-10.6±3.25	0.729
MI	-11 to -17	-15	-13.71±4.84	-11 to -19	-14	-12.31±4.84	0.381
MIL	-14 to -19	-17	-14.46±4.28	-12 to -22	-17	-13.96±4.28	0.749
MAL	-16 to -23	-18	-12.78±3.35	-10 to -23	-16	-10.38±3.35	0.780
AA	-17 to -20	-20	-8.38±4.07	-14 to -20	-18	-13.58±4.04	0.813
AS	-14 to -27	-20	-17.2±6.79	-15 to -23	-17	-14.21±6.79	0.858
AI	-15 to -32	-20	-16.71±0.73	-14 to -20	-18	-16.79±3.32	0.488
AL	-15 to -28	-20	-17.11±5.27	-11to -22	-20	-15.31±5.27	0.802
A	-11 to -20	-17	-12.06±3.80	-11to -20	-17	-13.06±3.80	0.700
GLS	-11 to -24	-14	-11.11±2.90	-9to -23	-13	-11.16±4. 66	0.817
All numericals in %							

Table 6 shows values for the range, mean and median segmental strain in both the groups. There were wide variations in segmental PSLS values in all regions. In both the groups, average PSLS values were less negative in basal and mid segments than apical segments which demonstrated more negative strain. There was no statistical difference found between the groups in pre CPB period. Abbreviations **:-** BA**:** basal anterior; BAS-basal anteroseptal, BIS-basal inferoseptal, BI-basal inferior, BIL-basal inferolateral, BAL-basal anterolateral, MA-mid anterior, MAS-mid anteroseptal, MIS-mid inferoseptal, MAL-mid anterolateral, AA-apical anterior, AS-apical septal, AI-apical inferior, A-apex, GLS-Global longitudinal strain.

Table 7 – Comparison of postoperative strain values (range, median and mean) between sevoflurane and propofol group

	S- Group			P- Group			P value
	Range (%)	Median (%)	Mean (%)	Range (%)	Median (%)	Mean (%)	
BA	-7 to -19	-15	-13.29±4.532	-10 to -16	-13	-14.08±5.10	0.421
BAS	-8 to -17	-15	-10.8±3.738	-8 to -17	-13	-11.47±3.33	0.136
BIS	-11 to -16	-14	-13.78±5.258	-9 to -16	-13	-11.41±5.24	0.121
BI	-9 to -17	-10	-13. 6 ±6.090	-8 to -19	-11	-17.08±.89	0.528
BIL	-7 to-21	-18	-15.86±6.00	-9 to -13	-13	-17.19±7.02	0.087
BAL	-11 to -18	-15	-13.954±.434	-14 to -20	-16	-15.75±.95	0.994
MA	-13 to -18	-15	-13.52±5.382	-7 to -21	-14	-13.47±4.91	0. 617
MAS	-15 to -23	-18	-15.5±7.704	-10 to -22	-14	-16.09±8.43	0.090
MIS	-16 to -20	-18	-13.39±5.451	-11 to -23	-16	-15.83±.27	0.320
MI	-16 to-19	-17	-16±4.811	-11 to -19	-17	-14.83±.42	0. 672
MIL	-16 to -23	-19	-14.041±.937	-13 to -24	-15	-17.9±4.75	0.459
MAL	-17 to -24	-19	-14.347±5.30	-11 to -26	-17	-15.5±5.85	0. 621
AA	-14 to -30	-23	-18.384±7.25	-16 to -23	-20	-19.12±8.55	0.210
AS	-15 to -28	-22	-18.391±7.58	-14 to -30	-20	-17.37±7.52	0.759
AI	-17 to-30	-17	-18.3±7.832	-17 to -22	-22	-18.55±7.75	0.561
AL	-21 to -30	-23	-16.458±7.15	-15 to -27	-22	-17.33±.81	0.532
A	-17 to -24	-25	-17.307±6.82	-16 to -23	-19	-17.58±.43	0.800
GLS	-12 to -21	-15	-13.28±4.53	-11 to -24	-14	-14.7±4.88	0.571
All numericals in %							

Table 7 shows the range, mean and median segmental strain values in both the groups. There were wide variations in segmental PSLS values in all regions. Average PSLS values were less negative in basal and mid segments than apical segments which demonstrated more negative strain. There was no statistical difference found between the groups in post- CPB period

Intra-group comparison of segmental PSLS between the pre-CPB and post-CPB period revealed that the frequency of segments with abnormal strain values in pre-CPB period showing increment in segmental PSLS values in post-CPB were equal between the groups except in apical cap.

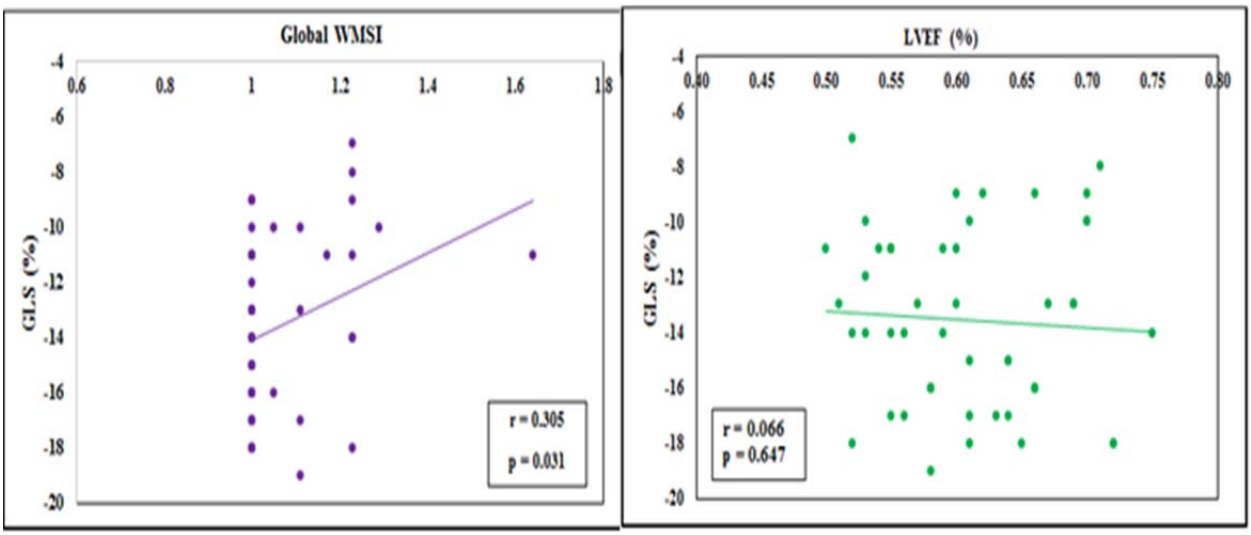
Table 8- Comparison of segmental strain values within the groups in pre-CPB and post-CPB period

Segment	**S –Group (n=26)**						**P-Group(n=24)**					
	Pre-CPB		Post-CPB			P value	Pre-CPB		Post-CPB			P value
	Nor	Ab	Nor	Ab	UD		Nor	Ab	Nor	Ab	UD	
BA	2	24	6	16	2	0.071	2	24	7	16	1	0.871
BAS	2	24	6	19	1	0.251	3	21	5	18	1	0.541
BIS	3	23	7	16	3	0.074	2	22	6	17	1	0.254
BI	2	24	9	15	2	0.27	4	20	16	7	1	0.736
BIL	9	17	14	10	1	0.117	9	15	10	11	3	0.067
BAL	2	24	8	16	2	0.979	4	20	12	12	0	0.346
MA	3	23	9	16	1	0.372	3	21	8	15	1	0.478
MAS	11	15	14	11	1	0.104	4	20	10	12	2	0.883
MIS	6	20	7	18	1	0.204	5	19	13	11	0	0. 672
MI	9	17	15	10	1	0.723	8	16	14	8	2	0.344
MIL	5	21	10	14	2	0.936	6	18	12	11	1	0.553
MAL	5	21	10	15	1	0.464	3	21	13	10	1	0.145
AA	5	21	11	15	1	0.069	7	17	14	6	4	0.322
AS	6	20	12	13	1	0.445	7	17	12	6	6	0.094
AI	8	18	16	9	1	0.283	11	13	17	6	1	0.556
AL	10	16	14	11	1	0.145	10	14	15	9	0	0.113
A	14	12	17	9	0	0.003	12	12	15	9	0	0.074

P value calculated for preoperative Ab to postoperative Nor values

Table 8 shows comparison of segmental PSLS in all regions between pre and post CPB period in both groups. The frequency of segments with abnormal strain in pre CPB period demonstrating normal strain values in post CPB were similar in both the groups except in Apex (A) in sevoflurane group which showed better improvement. Abbreviations:- UD : undetermined : Nor: Normal, Ab- abnormal

Global WMSI, LVEF were compared with GLS using Pearson's correlation in all 50 patients in pre-CPB period. There was weak positive correlation between WMSI and GLS (r=0.305), but there was no correlation between GLS and LVEF (Figure 5).

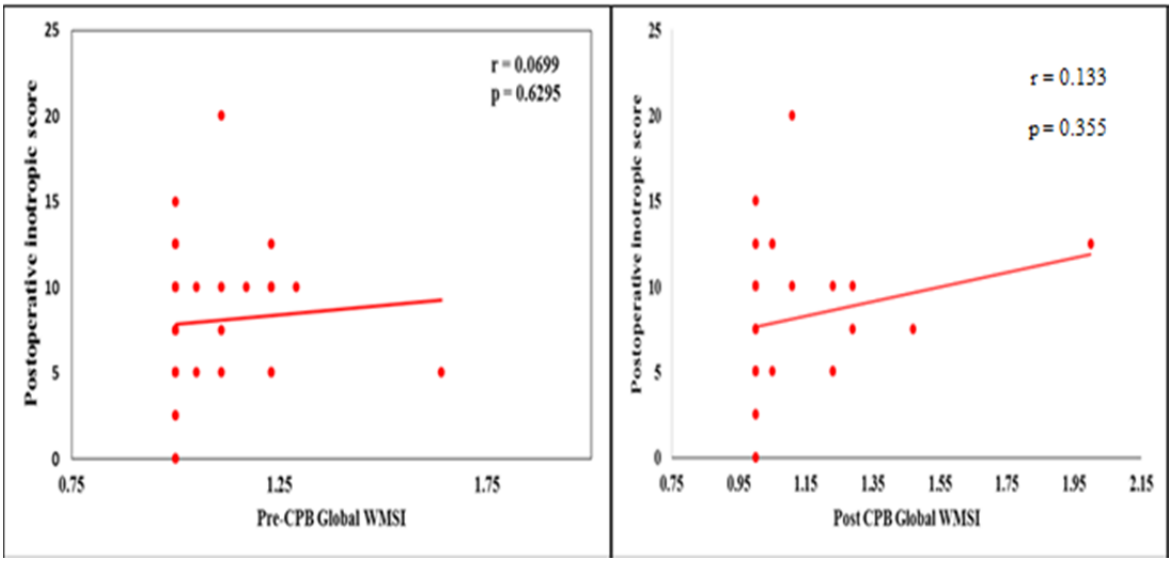


A. **B.**

Figure 5:- Correlation between global WMSI, LVEF with GLS is shown in the figure. (A) The correlation of GLS versus WMSI in all patients revealed a direct weak correlation. (B) There was no correlation between LVEF and GLS.

Pearson's correlation co-efficient (r) - positive value denotes direct correlation whereas negative value signifies inverse correlation. (0 to 0.35- poor/weak correlation, 0.35 to 0.55- good correlation , >0.55- significant correlation).

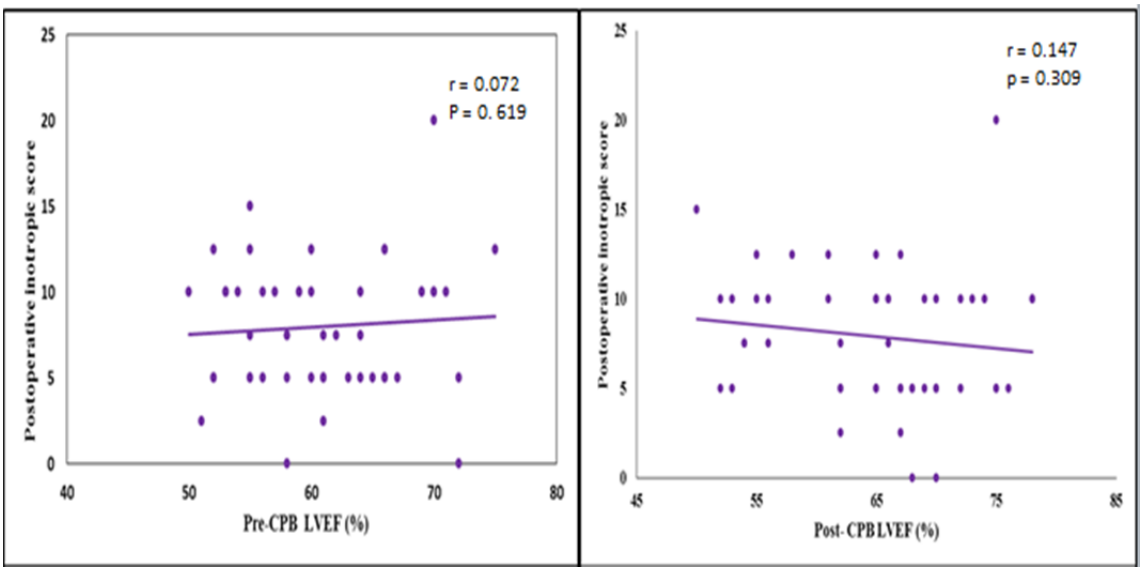
Pre-CPB and post-CPB WMSI was correlated with postoperative inotropic score. There was poor direct correlation between pre-CPB WMSI with post-operative inotropic score (r=0.0699). Similar (r=0.133) was noted between post-CPB WMSI and postoperative inotropic score (Figure- 6).



A. B.

Figure 6 – Pearson's correlation is seen between WMSI in both pre-CPB and post-CPB period with postoperative inotropic score. (A) There was poor direct correlation between pre-CPB WMSI with postoperative inotropic score (r=0.0699). (B) Similar correlation (r=0.133) was noted between post-CPB WMSI and postoperative inotropic score

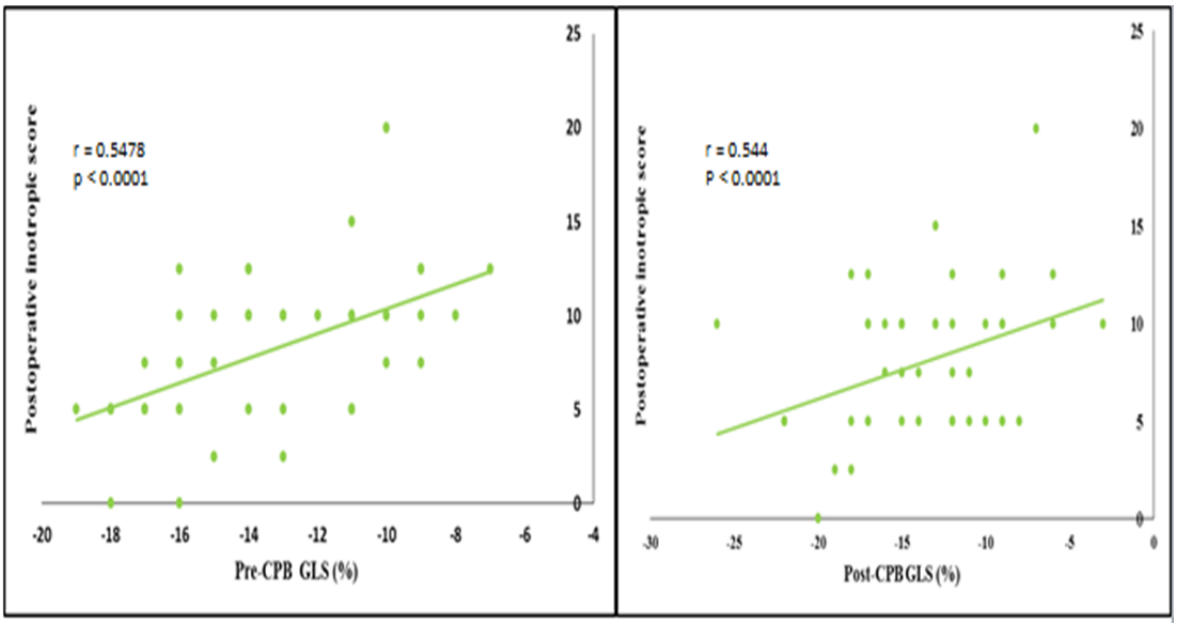
LVEF in pre-CPB and post-CPB period (Figure 7) showed no correlation with inotropic requirement in postoperative period (r=0.072 and r=0.147 respectively).



A. **B.**

Figure 7- Pearson's Correlation between LVEF and Postoperative inotropic score. LVEF showed a poor correlation with postoperative inotropic score in pre-CPB (A) and post-CPB (B) period.

GLS values obtained in pre and post-CPB period demonstrated a good direct correlation with inotropic score in postoperative period i.e. with less negative strain values there was increase in need for inotropes in both the groups (Figure 8).



A. **B.**

Figure 8- Pearson's Correlation between GLS and postoperative inotropic score. (A). In pre –CPB period GLS showed a good positive correlation with requirement of inotropes in postoperative period. (B) Correlation was good between post-CPB GLS and postoperative inotropic score.

DISCUSSION

DISCUSSION

In this prospective, randomized study, we compared the effects of sevoflurane and propofol at equi-anesthetic doses (to maintain BIS value in the range of 40-60) on segmental PSLS and GLS in patients undergoing CABG. Patients having isolated CAD in whom TTE demonstrated LVEF >55% were included as study subjects. Patients received either sevoflurane or propofol throughout the intraoperative period titrated to BIS value of 40-60. Other factors affecting myocardial function like fentanyl dosage, CPB duration, aortic cross clamp time, no. of bypass grafts were constant during the study period. Since strain is affected by loading condition, effects of these agents on myocardial strain were assessed at constant loading conditions, maintaining baseline values for CVP, SBP & MAP in both groups within ±20% of the limits. Conventional echocardiographic parameters (2D Biplane LVEF, SVI, CI, WMSI), segmental PSLS, GLS and post-operative inotropic score were compared between the groups in both pre and post-CPB period. Also, all the above parameters were compared in pre-CPB and post-CPB period within the group.

Many factors are known to influence the perioperative LV systolic function during CABG surgery. These include patient characteristics (age, extent of coronary artery disease and degree of underlying LV dysfunction etc.) and surgery related events such as number and quality of grafts, type of cardio-protection, duration of aortic cross-clamp and CPB. In our study, patient characteristics and surgical features were similar in both the groups. This suggests that the differences in cardiac function between the groups were not associated with patient's characteristics and intraoperative events but with the choice of anesthetic agents. Even though, opioids have been shown to offer cardio protective effect similar to that of ischemic preconditioning[61], we do not attribute the

observed differences in cardiac function between the groups to fentanyl as it was administered in similar dosage in both the groups.

Various studies have reported utility of conventional echocardiographic techniques for the evaluation of effects of sevoflurane and propofol on LV global and regional systolic function. The commonly employed intraoperative method of WMSI that quantifies regional myocardial function has many limitations- 1) It is highly subjective, operator-dependent and scoring is based upon qualitative evaluation of wall motion. 2) It focuses only on the function of radial myocardial fibres without taking into account the functions of longitudinal fibres, which are the first one to get involved at early stages of myocardial ischemia/ dysfunction. Even LVEF, a frequently used quantitative parameter of LV systolic function is affected only when significant number of myocardial segments becomes dysfunctional. Hence conventional methods of echocardiography do not provide insight into the contractile dysfunction of different categories of myocardial fibres. On the contrary, the strain quantification is more valuable for the evaluation of different types of LV contractile forces.

Currently, myocardial strain quantification can be performed using methods such as - TDI imaging and STE. Due to angle-dependency, the TDI-strain quantification has limitations in interrogating the mid and apical segments of the LV. Unlike TDI, the STE strain measurement being angle-independent can quantify the strain values more accurately. It calculates deformation in two axes and can measure strain simultaneously in the longitudinal and transverse direction in long-axis views, and radial and circumferential direction in short-axis views. Hence, STE is capable of quantifying regional deformation in all the myocardial segments. Also, it is less affected by tethering or translation artifacts and can distinguish lack of active contraction from passive motion. So, STE strain provides robust measurements of myocardial deformation with

acceptable intra-observer and inter-observer variability. Therefore, strain quantification may be regarded as a reference method for the assessment of regional myocardial function although it is more time-consuming than the routine wall motion analysis. Our study has shown that the regional and global strain quantification using STE is more sensitive in detecting subtle changes in the LV contractility compared to the conventional 2D echocardiographic methods such as WMSI and LVEF in patients undergoing CABG under either sevoflurane or propofol anesthesia.

The main findings of this prospective study are

1. The feasibility of segmental PSLS was 99% and 92% in pre and post- CPB period respectively.

2. In both sevoflurane and propofol group there were wide variations in values of segmental strain in any individual myocardial segment in different patients.

3. The hypoperfused segments defined angiographically exhibited normal as well as abnormal PSLS values in different patients.

4. Segmental PSLS and GLS values were comparable in all patients both in sevoflurane and propofol group during pre-CPB period. Similar results were obtained between the groups in post-CPB period.

5. The median and mean±sd of GLS values were -14% and -11.11±2.90% in sevoflurane group and -13% and -11.16±4.66% in propofol group in the pre-CPB period. In post-CPB period, the median and mean±SD GLS values (with inotropes) were -15% and -13.28±4.28% and -14% and -14.7±4.88% in sevoflurane and propofol group respectively.

6. The frequency of segments demonstrating incremental values in segmental strain (abnormal turning to normal) during post-CPB period was similar in both the groups. (Sevoflurane 21.11% vs. Propofol 24%)

7. The average inotropic score was not statistically significant between the groups.

8. In both the groups, only GLS showed good positive correlation with post-operative inotropic requirements. There was weak correlation between conventional echocardiographic parameters (LVEF, SVI, CI, and WMSI) and post-operative inotropic score.

9. WMSI and LVEF have poor positive linear correlation with GLS.

Feasibility of Segmental and global strain in pre-CPB and post-CPB period

In our study, Strain imaging was feasible in all the included patients in pre-CPB period but, 20 patients were excluded due to poor TEE images, arrhythmias (VPC's, atrial fibrillation) and electro cautery disturbances during post-CPB period, wherein strain analysis was not possible. Even though several studies[33, 43] have reported 99% feasibility of segmental strain imaging during cardiac surgery, in our study during post-CPB period strain quantification could be performed in 92% of the segments. The STE strain quantification depends on the quality of the 2D images recorded in three ME views. Interpretation of the speckle tracking is largely influenced by the frame rate, gain settings, accuracy in tracing endocardial borders and the movement of the myocardium. The increasing myocardial motion because of administration of inotropes and tissue edema in the post-CPB period deteriorates the image quality and strain analysis which was evident in our patients.

Effect of sevoflurane and propofol on hemodynamics

The sevoflurane was administered in the concentration of 0.8-1.2 MAC whereas the propofol was infused at a dose range of 75-100μg/kg/min. Propofol is known to produce hypotension in patients with poor LV function. Since, the patients in our study group had good LV function we excepted occurrence of less hemodynamic disturbances in both the groups. Administration of large doses of fentanyl in propofol group may be associated with bradycardia if the patient receives adequate volume replacement. However, we used moderate doses of fentanyl (5-10μg/kg) during induction. Hence, average heart rate in pre CPB period was comparable to baseline heart rate in both the groups. In post CPB period, there was parallel surge in average heart rate in both groups when compared with baseline values. This can be attributed to use of vasoactive agents (adrenaline) in both the groups.

Average MAP in pre-CPB period was reduced in both the groups when compared to baseline. The reduction in MAP during pre-CPB was significant in propofol group ($p=0.013$) than sevoflurane group. Nishikawa et al [34] showed similar reduction in MAP in sevoflurane and propofol group. The incidence of hypotension in our patients was transient and could be easily managed with titration of anesthetic agents, fluid replacement and injection of boluses of vasopressors in both the groups. More number of patients in propofol group had variable hemodynamic response in pre-CPB period compared to sevoflurane. Some of the patients had transient episodes of hypotension whereas some of the other patients had hypertensive responses requiring nitroglycerin to maintain MAP under control. Our finding is consistent with those of Gravel et al[62] who reported higher incidence of hypertension in propofol group before establishing CPB. However, the MAP values didn't differ from baseline values after the patients were weaned from CPB.

Effect of sevoflurane and propofol on conventional echocardiographic parameters (WMSI, SVI, CI and LVEF)

Both sevoflurane and propofol produce hemodynamic disturbances when administered in high concentration. Arterio-venodilatation is known to occur after propofol infusion. Large number of studies has shown beneficial effects of sevoflurane in preserving the myocardial function [60, 61]. As a consequence, both the agents may have variable effect on myocardial function which may be reflected on conventional echocardiographic parameters. However, no substantial differences were found on conventional echocardiographic variable between the groups in our patients. Our findings are consistent with those published by Lindholm et al[26]. There was significant increase in CI, LVEF and SVI after CPB in both the groups which may be attributed to inotropic and chronotropic action of vasoactive agents. However, the magnitude of increase in these parameters remained similar in both the groups. WMSI did not alter from pre-CPB to post-CPB period despite administration of inotropes in both the groups.

Some of the researchers during their study found that echocardiographically derived cardiac index was more and troponin level were less in sevoflurane group compared to propofol group after revascularization in patients operated for CABG[24,63]. These changes they attributed to the cardio-protective effects of sevoflurane. Sevoflurane was also shown to reduce the ischemia-induced metabolic changes in myocardium despite hypotension which were not seen with the propofol.

Effect of sevoflurane and propofol on segmental peak systolic longitudinal strain (PSLS) and GLS in CAD patients.

The values for segmental PSLS do not remain constant in all myocardial segments, but increase from base to apex. This finding also has been reported by Sutherland et al[11] and

Voigt et al[39]. We also noted wide variations in absolute values for segmental strain in both the groups. Sutherland et al [11] mentioned that the longitudinal systolic peak strain is often (but not always) marginally higher in the mid segments than in the basal segments in all 4 cardiac walls. The ischemic segments of myocardium can be identified based on angiographic findings. However angiographic definition of ischemia based on coronary stenosis may not be synonymous with reduced perfusion as the distal segments may derive perfusion through collateral vessels. Angiographically defined ischemic segments predominantly revealed abnormal strain values (less negative values) but, a significant number of these segments also displayed normal strain (PSLS>-17). This is in contrast to previous publication by Kukucha et al[43] who reported that strain analysis could detect wall motion difference between normally perfused and ischemic segments. We attribute inability to distinguish normally perfused segments from ischemic segments in our subset of patients to the lack of consideration to stenosis in distal intramyocardial vessels and possible presence of coronary collaterals. Our findings suggest that even strain analysis can't differentiate normally perfused and ischemic segments but a large scale studies are required to confirm this findings. The percentage of segments with low PSLS, which displayed incremental strain values in the post CPB period, was similar in both the groups. This suggests that both sevoflurane and propofol didn't differ in terms of myocardial protection. This finding is in agreement with the study conducted by Lindholm et al [26] who reported that there was no obvious advantage of sevoflurane over TIVA method in improving the indices of cardiac function. Whether the depth of anesthesia alters the strain quantification still remains a matter to be investigated. Even though, the BIS value in our patients was lower down to 40-60 in the intraoperative period, this depth of anesthesia cannot be ascribed to the individual anesthetic effects of sevoflurane or propofol alone, but may be attributed to the combined effect of both

inhaled and intravenously administered anesthetic drugs. Hence, the amount of sevoflurane or propofol administered to the patients probably was not large enough to induce changes in the PSLS. It has been demonstrated in animal studies [45, 46] that STE parameters are significantly reduced only under deep anesthesia. About 45 out of 50 of our patients received inotropes in small dose (adrenaline or nor-adrenaline not exceeding 0.05μg/kg/min) in post-CPB period. Even though choice and dosage of these inotropes may affect contractility and vasoconstriction, no significant difference was observed between the groups and also between the pre-CPB and post-CPB period. This may be explained by the use of relative low and equal doses of inotropes in both groups. Besides, all our patients had good LV to begin with: hence, probably there was marginal improvement in values of PSLS with the administration of inotropes.

Correlation of LVEF, WMSI, CI and GLS with post-operative inotropic score

Twenty one out of 26 patients in sevoflurane group and all patients in propofol group were given inotropic or vasoconstrictive support after CPB and in first few hours in intensive care unit. The mean inotropic score was 7.50±4.02 in sevoflurane group .and 8.54±3.529 in propofol group. The need for post- operative inotropic score was best predicted by GLS in pre-CPB period i.e. patients having less negative strain values required more inotropes. Conventional 2D echocardiography parameters showed no correlation with inotropic score. These findings are consistent with the previous published study, which showed that preoperative GLS has potential to predict prolonged inotropic requirement after cardiac surgery[64]. In post-CPB period, inotropic treatment might have influenced the analysis of segmental and global strain in both the groups. Hence, the values obtained in post-CPB period may not be attributed to the net effects of sevoflurane or propofol on strain parameters.

Limitations of our study

1. Sample size and power calculation in the published reference study was based on troponin release and not on echocardiographic data. However, due to lack of studies on the effects of anesthetic agents on strain quantification, we performed sample sizing based on the reference study.

2. Given the small sample size of our cohort, further large studies are required to validate these findings

3. Due to technological limitations the strain quantification was performed offline in our study, unlike the routine parameters such as LVEF, WMSI and CI that could be derived in the intraoperative period. With technological advances in future, it may become feasible to perform live strain quantification.

4. The system for WMS is well established and categorizes the wall motion into 5 grades. Due to the insufficient available data, no reference values are established for categorizing the normal and ischemic myocardial segments using strain quantification in the intraoperative period.

5. As the technology of strain quantification remains vendor-specific, there is no consensus among the vendors on strain analysis. Our strain analysis was performed in Philips Q-lab software which may not be same with software provided by other vendors. Quantification of strain in images moving out of plane remains a problem.

CONCLUSIONS

CONCLUSIONS

1. The present study suggests that there is no difference between sevoflurane versus propofol on myocardial contraction mechanics as studied using 2D echocardiographic strain imaging in low-risk patients undergoing CABG and having good baseline LV function.

2. We conclude that there are no significant differences in segmental PSLS and GLS between patient's anesthesized with sevoflurane or propofol in the pre-CPB and post-CPB period.

3. Our study demonstrated that there is no correlation between wall motion score index and global longitudinal strain.

4. The post-operative inotropic requirement was best predicted by GLS in patients having less negative value, requiring more inotropes during post bypass.

5. On comparing the effects of sevoflurane and propofol on conventional 2D echocardiographic parameters of LV systolic function (Wall motion score, Ejection fraction by Simpson's method, LVOT Cardiac output), we found that there is no difference between the agents in altering these parameters both in pre-CPB and post-CPB period.

BIBILOGRAPHY

BIBILOGRAPHY

1. Ascione R, Narayan P, Rogers CA, Lim KH, Capoun R, Angelini GD. Early and midterm clinical outcome in patients with severe left ventricular dysfunction undergoing coronary artery surgery. Ann Thorac Surg. 2003; 76:793-9.

2. Roques F, Nashef SA, Michel P et al .Risk factors and outcome in European cardiac surgery: Analysis of the Euro SCORE multinational database of 19030 patients. Eur J Cardiothorac Surg. 1999;15:816–23.

3. Hamad MA, Van Straten AH , Schönberger JP, et al. Preoperative ejection fraction as a predictor of survival after coronary artery bypass grafting: Comparison with a matched general population. J Cardiothorac Surg 2010;5:29.

4. Nesbitt GC, Mankad S, Oh JK. Strain imaging in echocardiography: methods and clinical applications. Int J Cardiovasc Imaging 2009; 25: 9–22.

5. Kalam K, Otahal P, Marwick TH. Prognostic implications of global LV dysfunction: a systematic review and meta-analysis of global longitudinal strain and ejection fraction. Heart 2014; 100: 1673–1680.

6. Klein P, Holman ER, Michel, Versteegh M, et al. Wall motion score index predicts mortality and functional result after surgical ventricular restoration for advanced ischemic heart failure. European Journal of Cardio-thoracic Surgery .2009 ;60 :847—853.

7. Sheehan FH. Quantitative evaluation of regional left ventricular systolic function. In: Otto CM eds. The Practice of Clinical Echocardiography. Philadelphia: WB Saunders Company 2002; 65- 87.

8. Force T, Kemper A, Perkins L, et al. Overestimation of infarct size by quantitative two-dimensional echocardiography. The role of tethering and of analytic procedures. Circulation 1986.73.1360- 68.

9. Chandra S, Skali H, Blankstein R- Novel techniques for assessment of left ventricular systolic function. Heart Fail Rev. 2010;16: 327-337.

10. Mor-Avi V, Lang RM, Badano LP, Belohlavek M, Cardim NM, Derumeaux G et al. Current and evolving echocardiographic techniques for the quantitative evaluation of cardiac mechanics: ASE/EAE consensus statement on methodology and indications endorsed by the Japanese Society of Echocardiography. Eur J Echocardiogr. 2011;12:167–205.

11. Sutherland GR, Di Salvo G, Claus P, et al: Strain and strain rate imaging: A new clinical approach to quantifying regional myocardial function. J Am Soc Echocardiogr. 2004; 17:788-802.

12. Nesbitt GC, Mankad S, Oh JK. Strain imaging in echocardiography: methods and clinical applications. Int J Cardiovasc Imaging 2009; 25: 9–22.

13. Hanekom L, Jenkins C, Jeffries L, et al. Incremental value of strain rate analysis as an adjunct to wall-motion scoring for assessment of myocardial viability by Dobutamine echocardiography: a follow-up study after revascularization. Circulation 2005; 112: 3892–3900.

14. Edvardsen T, Skulstad H, Aakhus S, Urheim S, Ihlen H. Regional myocardial systolic function during acute myocardial ischemia assessed by strain Doppler echocardiography. J Am Coll Cardiol 2001; 37: 726–730.

15. Reimer KA, Lowe JE, Rasmussen MM, Jennings RB. The wavefront phenomenon of ischemic cell death. 1. Myocardial infarct size vs duration of coronary occlusion in dogs. Circulation 1977;56:786-94.

16. Stadnicka A, Marinovic J, Ljubkovic M, Bienengraeber MW, Bosnjak ZJ. Volatile anesthetic-induced cardiac preconditioning. J Anesth. 2007;21(2):212–9.

17. Landoni G, Biondi-Zoccai GG, Zangrillo A, Bignami E, D'Avolio S, Marchetti C, et al. Desflurane and sevoflurane in cardiac surgery: a meta-analysis of randomized clinical trials. J Cardiothorac Vasc Anesth. 2007;21(4):502–11

18. Symons J, Myles P. Myocardial protection with volatile anaesthetic agents during coronary artery bypass surgery: a meta-analysis. Br J Anaesth. 2006;97(2):127–36.

19.Yu CH, Beattie WS. The effects of volatile anesthetics on cardiac ischemic complications and mortality in CABG: a meta-analysis. Can J Anaesth. 2006;53(9):906–18.

20.Landoni G, Greco T, Biondi-Zoccai G, Neto CN, Febres D, Pintaudi M, et al. Anaesthetic drugs and survival: a Bayesian network meta-analysis of randomized trials in cardiac surgery. Br J Anaesth. 2013;111(6):886–96.

21. Joo HS, Perks WJ. Sevoflurane versus propofol for anesthetic induction: a meta-analysis. Anesthesia & Analgesia. 2000;91(1):213–9.

22. Jakobsen C-J, Berg H, Hindsholm KB, Faddy N, Sloth E. The influence of propofol versus sevoflurane anesthesia on outcome in 10,535 cardiac surgical procedures. J Cardiothorac Vasc Anesth. 2007;21(5):664–71.

23. Yao Y-T, Li L-H. Sevoflurane Versus Propofol for Myocardial Protection in Patients Undergoing Coronary Artery Bypass Grafting Surgery: a Meta-analysis of Randomized Controlled Trials. Chin Med Sci J. 2009;24(3):133–41

24. De Hert SG1, Cromheecke S, Ten Broecke PW, Mertens E, De Blier IG, Stockman BA et al. Effects of propofol, desflurane, and sevoflurane on recovery of myocardial function after coronary surgery in elderly high-risk patients. Anesthesiology. 2003;99(2):314-23

25. Bharti N, Chari P, K S Thingnam, Arora S. Comparison of Hemodynamic and Cardiovascular effects of VIMA with Sevoflurane Versus TIVA with Propofol in Patients Undergoing Coronary Artery Bypass Surgery. Indian Journal of Anaesthesia. 2008; 52 (6):805-812

26. Lindholm E E , Aune E, Froland G, Kirkeben KA, Otterstad JE. Analysis of transthoracic echocardiography data in major vascular surgery from a prospective randomized trial comparing sevoflurane and fentanyl with propofol and remifenatnyl anesthesia. Anaesthesia .2014;69 :6.

27. Braunwald E. Heart failure. JACC Heart Fail 2013; 1:1—20.

28. Cheitlin MD, Armstrong WF, Aurigemma GP, et al. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography- summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). J Am Coll Cardiol. 2003; 42:954-70.

29. Marwick TH. Should we be evaluating the ventricle or the myocardium? Advances in tissue characterization. J Am Soc Echocardiogr. 2004;17:168-72.

30. Chandra S, Skali H, Blankstein R: Novel techniques for assessment of left ventricular systolic function. Heart Fail Rev.2010; 16:327-337.

31. Palmieri V, Russo C, Buonomo A, Palmieri EA, Celentano A. Novel wall motion score-based method for estimating global left ventricular ejection fraction. Validation by real-time 3D echocardiography and global longitudinal strain. European Journal of Echocardiography.2010; 11:125-130.

32. Comunale ME, Body SC, Ley C, Koch C, Roach G. The concordance of intraoperative left ventricular wall motion abnormalities and electrocardiographic ST-segment changes: association with outcomes after coronary revascularization.Anesthesiology.1998; 88:945-954.

33. Menting M, McGhie JS, Koopman LP, Vletter WB, Helbing WA. Normal myocardial strain values using 2D speckle tracking echocardiography in healthy adults aged 20 to 70 years. Echocardiography.2016;33, 1665-1675.

34. Nishikawa k, Kanayab N, Kawamata M, Namiki A. Left ventricular mechanical performance in elderly patients after induction of anesthesia. A comparison of inhalational induction with sevoflurane and intravenous induction with fentanyl and propofol. Anesthesia 2004, 59, 948-953.

35. Thomas JD, Popovic ZB. Assessment of left ventricular function by cardiac ultrasound. J Am Coll Cardiol.200,48, 2012-25.

36. Leitman M, Lysyansky P, Sidenko S, Shir V, Peleg E, Binenbaum M, et al. Two-dimensional strain- a novel software for real- time quantitative echocardiographic assessment of myocardial function. J Am Soc Echocardiogra. 2004; 17 : 1021-9.

37. Duncan A, Alfirevic A , Sessler D, Popovic ZB, Thomas JD. Perioperative assessment of myocardial deformation. Anesth Analg. 2014; 118: 524-544.

38. Geyer H, Caraccibo G, Wilansky S, Carerj S, Gentile F et al. Assessment of myocardial mechanics using speckle tracking echocardiography. Fundamentals and clinical applications. J Am Soc Echocardiogr. 2010 ; 23: 351-9. Erratum in J Am Soc Echocardiogr. 2010; 23: 734

39. Jens-Uwe Voigt, Gianni Pedrizzetti,Peter Lysyansky, Tom H. Marwick,Helen Houle, Rolf Baumann et al. Definition for a common standard for 2D speckle tracking echocardiography : consensus document of the EACVI/ASE/Industry Task force to standardize deformation imaging. European heart journal- Cardiovascular imaging 2015; 16:1-11.

40. Brown J, Jenkins C, Marwick TH. Use of myocardial strain to assess global left ventricular function: a comparison with cardiac magnetic resonance and 3-dimensional echocardiography. Am Heart J. 2009; 157(1):102.

41. Aurich M, Keller M, Greiner S, Steen H, Aus dem Siepen F, Riffel J, Katus HA et al. Left ventricular mechanics assessed by two-dimensional echocardiography and cardiac magnetic resonance imaging: comparison of high-resolution speckle tracking and feature tracking. Eur Heart J Cardiovasc Imaging. 2016 Dec; 17:1370-78.

42. Jin-Oh Choi, Sung Won Cho, Young Bin Song, Soo Jin Cho, Bong Gun Song, Sang-Chol Lee. Longitudinal 2D strain at rest predicts the presence of left main and three vessel coronary artery disease in patients without regional wall motion abnormality. European Journal of Echocardiography 2009; 10: 695–701.

43. Kukucka M, Nasseri B, Tscherkaschin A, Mladenow A, Kuppe M, Habazettl H. The Feasibility of Speckle Tracking for Intraoperative Assessment of Regional Myocardial Function by Transesophageal Echocardiography .Journal of Cardiothoracic and Vascular Anesthesia. 2009:23; 462-467.

44. Adamo L, Perry A, Novak E, Makan M, Brian R. Lindman, Douglas L. Mann. Abnormal Global Longitudinal Strain Predicts Future Deterioration of left Ventricular Function in Heart Failure Patients with a Recovered left Ventricular Ejection Fraction. Circ Heart Fail. 2017; 10: e003788.

45. Wang G, Zhang L, Zhang C. Effects of anesthesia on conventional and speckle tracking echocardiographic parameters in a mouse model of pressure overload. Exp Ther Med. 2015;9: 1679-87.

46. Ann-Sabin B, Schefer RJ, Schwarzwald. The use of strain, strain rate and displacement by 2D speckle tracking for assessment of systolic left ventricular function in goats, applicability and influence of general anesthesia. Cardiovasc Ultrasound. 2015;13: 11.

47. Jakobsen CJ, Berg H, Hindsholm KB, Faddy N, Sloth E. The influence of propofol versus sevoflurane anesthesia on outcome in 10,535 cardiac surgical procedures. J Cardiothorac Vasc Anesth 2007; 21: 664–71

48. Landoni G, Biondi-Zoccai GG, Zangrillo A, et al. Desflurane and sevoflurane in cardiac surgery: a meta-analysis of randomized clinical trials. J Cardiothorac Vasc Anesth 2007; 21: 502–11.

49. Yu CH, Beattie WS. The effects of volatile anesthetics on cardiac ischemic complications and mortality in CABG: a meta-analysis. Can J Anaesth 2006; 53: 906–18

50. Symons JA, Myles PS. Myocardial protection with volatile anaesthetic agents during coronary artery bypass surgery: a meta-analysis. Br J Anaesth 2006; 97: 127–36.

51. Lurati Buse GA, Schumacher P, Seeberger E, et al. Randomized comparison of sevoflurane versus propofol to reduce perioperative myocardial ischemia in patients undergoing noncardiac surgery. Circulation 2012; 126: 2696–704.

52. Joo HS, Perks WJ. Sevoflurane versus propofol for anesthetic induction: a meta-analysis. Anesthesia & Analgesia. 2000 ;91(1):213–9.

53. Jakobsen C-J, Berg H, Hindsholm KB, Faddy N, Sloth E. The influence of propofol versus sevoflurane anesthesia on outcome in 10,535 cardiac surgical procedures. J Cardiothorac Vasc Anesth. 2007; 21(5):664–71.

54. Jakobsen CJ, Torp P, Vester L, Folkersen, Thougaard A , Sloth E. Ketamine reduce left ventricular systolic and diastolic function in patients with ischemic heart disease. Acta Anaesthesiol Scand 2010, 54, 1137-44.

55. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al.; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18(12):1440-63.

56. Kapoor PM, Chowdary U, Mandal B, Kiran U, Karnatak R. Transesophageal echocardiography in off-pump coronary artery grafting. Ann Card Anaesth 2009; 12 :167.

57. Montgomery DE, Puthumana JJ, Fox JM, Ogunyankin KO. Global longitudinal strain aids the detection of non-obstructive coronary artery disease in the resting echocardiogram. Eur Heart J Cardiovasc Imaging 2012; 13: 579–587.

58. Wernovsky G1, Wypij D, Jonas RA, Mayer JE Jr, Hanley FL, Hickey PR et al.Postoperative course and hemodynamic profile after the arterial switch operation in neonates and infants. A comparison of low-flow cardiopulmonary bypass and circulatory arrest. Circulation. 1995 Oct 15;92(8):2226-35

59. Yamazaki y, Oba K, Matsui Y, Morimoto Y. Vasoactive-inotropic score as a predictor of morbidity and mortality in adults after cardiac surgery with cardiopulmonary bypass. Journal of Anesthesia .2018,32,167-73.

60. De Hert SG, Cromheecke S, ten Broecke PW, Mertens E, De Blier IG, Stockman BA et al. Effects of propofol, desflurane, and sevoflurane on recovery of myocardial function after coronary surgery in elderly high-risk patients. Anesthesiology, 2003:99;314-23.

61.Sigg DC, Coles JA Jr, Gallagher WJ, Oeltgen PR, Iaizzo PA. Opioid preconditioning. Myocardial function and energy metabolism. Ann Thorac Surg, 2001, 72, 1576-82.

62. Gravel NR, Searle NR, Taillefer J, Carrier M, Roy M, Gagnon L. Comparison of the hemodynamic effects of sevoflurane anesthesia induction and maintenance vs TIVA in CABG surgery. Can J Anaesth. 1999 Mar;46(3):240-6.

63.Yu CH, Beattie WS. The effects of volatile anesthetics on cardiac ischemic complications and mortality in CABG: a meta-analysis . Can J Anaesth 2006; 53: 906–18

64. Ternacle J, Berry M, Alonso E, Kloeckner M, Couetil JP, Rande JLD, et al. Incremental value of global longitudinal strain for predicting early outcome after cardiac surgery .European Heart Journal – Cardiovascular Imaging .2013; 14 : 77–84.

ANNEXURE

Consent form

Title of the study:

TITLE: “Effect of sevoflurane versus propofol on segmental and Global longitudinal strain in patients undergoing coronary artery bypass surgery”

Study numbers: We request you to participate in the study wherein we are planning to evaluate the effects of sevoflurane vs propofol anesthesia on post-operative outcomes in patients undergoing coronary artery bypass grafting using intraoperative echocardiographic global longitudinal strain. We hope to include 50 people from this hospital in this study.

What is sevoflurane or propofol anesthesia?

Sevoflurane is an inhalational anesthetic agent used commonly in maintenance of general anesthesia. It will be delivered in appropriate concentration using anesthesia machine through endotracheal tube.

Propofol is an intravenous anesthetic used commonly for induction and maintenance of general anesthesia.

What is Trans-esophageal echocardiography?

Trans-esophageal echocardiography or TEE is a test that uses sound waves to create high-quality moving pictures of the heart and its blood vessels. This can pin point the problematic areas of the heart and helpful in assessing function of heart valves before and after valve replacement surgery. Usually echocardiography is performed via chest (transthoracic echocardiography). However, under anesthesia, transthoracic echocardiography cannot be performed during cardiac surgery as the surgeon will be operating in that area. Hence echocardiography probe is inserted via esophagus (food pipe). TEE involves a flexible tube (probe) with a transducer at its tip. The Anesthesiologist will guide the probe down your throat and into your esophagus (the passage leading from your mouth to your stomach). This will be done when you will be unconscious under anesthesia and it will not cause any discomfort to you. This approach allows your doctor to get more detailed pictures of your heart because the esophagus is directly behind the heart.

What is the role of global longitudinal strain?

Global longitudinal strain is a new technique where the images acquired during routine TEE will be analyzed offline in computer to quantify about myocardial function objectively

How is Transesophageal echocardiography performed?

This will be done when you are under anesthesia and will not cause any discomfort to you. Your Anesthesiologist will insert the lubricated probe into your mouth. He or she

will then gently guide it down your throat into your esophagus. Your esophagus lies directly behind your heart. During this process, Anesthesiologist will take care to protect your teeth and mouth from injury.

What are the risks and side-effects?

We do not expect that our study will cause any injury to you because our study protocol is a part of routine intraoperative TEE examination. You will be under the effect of anesthesia while the test is being performed, thus you are unlikely to experience any discomfort.

Why are we doing this study?

The purpose of this study is to assess the usefulness of global longitudinal strain for predicting post-operative outcomes in patients undergoing CABG with propofol or sevoflurane anesthesia. It will also help to explore further its utility during cardiac surgery.

Can you withdraw from this study after it starts?

Your participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If you do so, this will not affect your usual treatment at this hospital in any way.

What will happen if you develop any study related injury?

We do not expect any injury to happen to you because of our study as it is a part of routine examination during the surgery.

Will you have to pay for the study? No.

Will your personal details be kept confidential?

Your personal details will be kept confidential. The result 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.

If you have any further questions, please ask:

Dr. Chennakeshavallu G N, Senior Resident, Department of Anaesthesia (Ph No: 9914207949)

Dr.Rupa Sreedhar, Professor and Head of Department of Anaesthesia.

Dr.Shrinivas V Gadhinglajkar, Professor, Department of Anaesthesia.

Dr.Prasant kumar Dash, Professor, Department of Anaesthesia.

Dr.Prashanth Bhaskar , Senior Resident, Department of Anaesthesia

Dr.Mala Ramanathan, Member Secretary, IEC & Additional Professor(study independent contact person, tel:04712524-234)

DECLARATION

I, _____,

Participant's name: Date of Birth / Age (in years)

Son / daughter of _____ (Please tick boxes) •

Declare that I have read the above information provide to me regarding the study:

TITLE: "Effect of sevoflurane versus propofol on segmental and Global longitudinal strain in patients undergoing coronary artery bypass surgery"

- 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 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: Name of Witness

Signature: 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.

Dr. Chennakeshavallu G N,

Senior Resident, CVTA,

SCTIMST.

.

കോൺസെന്റ് ഫോം

ടൈറ്റിൽ ഓഫ് ദി സ്റ്റഡി:

ഗ്ലോബൽ ലോങ്ങിട്ടിനാൽ സ്ട്രെയിൻ ഫോർ ഇവള്അഷൻ ഓഫ് പോസ്റ്റ് ഓപ്പറേറ്റീവ് ഔറ്കോമേ ഇൻ പടൈന്റ്സ് ഉണ്ടർഗോയിങ് കൊറോണറി ആർട്ടറി ബിപാസ്സ് ഗ്രാഫ്റ്റിങ് റേസിവിങ് ഈതെർ സെവോഫ്ലുരനെ ഓർ പ്രൊപോഫോൾ അനസ്തേഷ്യ

സ്റ്റഡി നമ്പര്: വെ റികുസ്റ്റ് യു ട്ടോ പാർട്ടിസിപ്പറ്റി ഇൻ ദി സ്റ്റഡി വൈരത്തിന് വെ ഏറെ പ്ലാനിംഗ് ട്ടോ ഇവള്അറെ ദി എഫക്ട് ഓഫ് സെവോഫ്ലുരനെ വേർസ്സ് പ്രൊപോഫോൾ അനസ്തേഷ്യ ഓൺ പോസ്റ്റ്-ഓപ്പറേറ്റീവ് ഔറ്കോംസ് ഇൻ പടൈന്റ്സ് ഉണ്ടർഗോയിങ് കൊറോണറി ആർട്ടറി ബിപാസ്സ് ഗ്രാഫ്റ്റിങ് യൂസിങ് ഇന്ഗ്രോപ്പറേറ്റീവ് എക്കോകാർഡിയോഗ്രാഫിക് ഗ്ലോബൽ ലോങ്ങിട്ടിനാൽ സ്ട്രെയിൻ . വെ ഹോപ്പ് ട്ടോ ഇണകളുടെ 50 പടൈന്റ്സ് ഫ്രം ദിസ് ഹോസ്പിറ്റൽ ഇൻ ദിസ് സ്റ്റഡി

വാട്ട് ഈസ് സെവോഫ്ലുരനെ ഓർ പ്രൊപോഫോൾ അനസ്തേഷ്യ ?

സെവോഫ്ലുരനെ ഈസ് ആൻ ഇന്ഹലാഷ അനസ്തെറ്റിക് അഗെന്റ് യൂസ്ഡ് കോമ്മൺലൈ ഇൻ മൈന്റൈനൻസ് ഓഫ് ജനറൽ അനസ്തേഷ്യ . ഇറ്റ് വിൽ ബി ഡെലിവെർഡ് ഇൻ അപ്പ്രോപ്രിയേറ്റ് കോൺസെൻട്രേഷൻ യൂസിങ് അനസ്തേഷ്യ മെഷീൻ ത്രൂ എൻഡോട്രക്കിൽ ട്യൂബ്

പ്രൊപോഫോൾ ഈസ് ആൻ ഇന്റ്റവെൻസ് അനസ്തെറ്റിക് യൂസ്ഡ് കോമ്മൺലൈ ഫോർ ഇൻഡക്ഷൻ ആൻഡ് മൈന്റൈനൻസ് ഓഫ് ജനറൽ അനസ്തേഷ്യ .

വാട്ട് ഈസ് ട്രാൻസ്-ഈസോഫഗെയ്ൽ എക്കോകാർഡിയോഗ്രാഫ്യ് ?

ട്രാൻസ്-ഈസോഫഗെയ്ൽ എക്കോകാർഡിയോഗ്രാഫ്യ് ഈസ് എ ടെസ്റ്റ് ദാറ്റ് അസീസ് സൗണ്ട് വേവ്സ് ട്ടോ ക്രീറ്റ ഹൈ-ക്വാളിറ്റി മൂവിങ് പിച്ടുരെസ് ഓഫ് ദി ഹാർട്ട് ആൻഡ് ഇത് ബ്ലഡ് വെസ്സൽസ് .ദിസ് ക്യാൻ പിന്-പോയിന്റ് ദി പ്രൊബ്ലെംഠിക് ആർഎസ് ഓഫ് ദി ഹാർട്ട് ആൻഡ് ഹെൽപ്ഫുൾ ഇൻ അസ്സീസ്സിങ് ഫങ്ക്ഷന് ഓഫ് ഹാർട്ട് വാൽവേസ് ബിഫോർ ആൻഡ് ആഫ്റ്റർ വാൽവ് റീപ്ലേസ്മെന്റ് സർജറി.യൂസുള്ള്യ എക്കോകാർഡിയോഗ്രാഫ്യ് ഈസ് പെർഫോർഡ് വയ ചേഷ്ട (ട്രാൻസ്തൊറാസിക് എക്കോകാർഡിയോഗ്രാഫ്യ്). ഹൗഎവെർ, അണ്ടർ അനസ്തേഷ്യ,ട്രാൻസ്തൊറാസിക് എക്കോകാർഡിയോഗ്രാഫ്യ് കാന്നോറ്റ് ബി പെർഫോർഡ് ഡ്യൂറിങ് കാർഡിയാക് സർജറി ആസ് ദി സുർജിയോൻ വിൽ ബി ഓപ്പറേറ്റിംഗ് ഇൻ ദാറ്റ് ഏരിയ .ഹെൻസ് എക്കോകാർഡിയോഗ്രാഫ്യ് പ്രോബ് ഈസ് ഇന്സേർട്ട് വയ ഈസോഫഗ്സ്(ഫുഡ് പൈപ്പ്).ട്രാന്സെസോഫഗെയ്ൽ എക്കോകാർഡിയോഗ്രാഫ്യ് ഇൻവോൾവ്സ് എ ഫ്ലെക്സിബിലെ ട്യൂബ് (പ്രോബ്)വിത്ത് എ ട്രാൻസ്ഡ്യൂസർ അറ്റ് ഇത് ടിപ്പ്.ദി അനെസ്തേസിയോളജിസ്റ് വിൽ ഗൈഡ് ദി പ്രോബ് ഡൌൺ യുവർ ത്രോത്

ആൻഡ് ഇൻട്രോ യുവർ ഈസോഫഗ്സ് (ദി പാസ്സേജ് ലീഡിങ് ഫ്രം യുവർ മൗത് ട്ടോ യുവർ സ്റ്റോമച്ച) . ദിസ് വിൽ ബി ടണ് വ്തെൻ യു വിൽ ബി ഉണക്കൻസിക്സ് അണ്ടർ അനസ്തേഷ്യ ആൻഡ് ഇറ്റ് വിൽ നോട ക്സ് എനി ഡിസ്കഫോട് ട്ടോ യു. ദിസ് അപ്രോച്ച് അള്ളോഡസ് യുവർ ഡോക്ടർ ട്ടോ ഗെറ്റ് മോർ ഡീറ്റൈൽഡ് പിച്ടുരെസ് ഓഫ് യുവർ ഹാർട്ട് ബെചൌസ് ദി ഈസോഫഗ്സ് ഈസ് ഡിറെക്ടല്യ് ബെതിന്ദ് ദി ഹാർട്ട്

വാട്ട് ഈസ് ദി റോൾ ഓഫ് ഗ്ലോബൽ ലോങ്ങിട്ടിനാൽ സ്ട്രെയിൻ ?

ഗ്ലോബൽ ലോങ്ങിട്ടിനാൽ സ്ട്രെയിൻ ഈസ് എ ന്യൂ ടെക്നിക് വ്തെരെ ദി ഇമേജസ് അക്വിർഡ് ഡ്യൂറിങ് റൂട്ടിനെ ട്രാൻസ് ഈസോഫേല് എക്കോകാർഡിയോഗ്രാഫ്യ് വിൽ ബി അനലൈസ്ഡ് ഓഫ്ലൈൻ ഇൻ കമ്പ്യൂട്ടർ ട്ടോ കാന്റിഫയ എബൌട്ട് മയോകാർഡിൽ ഫങ്ക്ഷന് ഒബ്ജെക്റ്റിവേലി.

ഹൌ ഈസ് ട്രാന്സെസോഫഗെയ്ൽ എക്കോകാർഡിയോഗ്രാഫ്യ് പെർഫോർഡ് ?

ദിസ് വിൽ ബി ടണ് വ്തെൻ യു ഏറെ അണ്ടർ അനസ്തേഷ്യ ആൻഡ് വിൽ നോട ക്സ് എനി ഡിസ്കഫോട് ട്ടോ യു . യുവർ അനെസ്തേസിയോളജിസ്റ് വിൽ ഇന്സേര്ട് ദി ലൂബ്രിക്കേറ്റഡ് പ്രോബ് ഇൻട്രോ യുവർ മൗത് .ഹി ഓർ ശേ വിൽ തേൻ ജന്റലി ഗൈഡ് ഇറ്റ് ഡൌൺ യുവർ ത്രോത് ഇൻട്രോ യുവർ ഈസോഫഗ്സ്.യുവർ ഈസോഫഗ്സ് ലിസ് ഡിറെക്ടല്യ് ബെതിന്ദ് യുവർ ഹാർട്ട് . ഡ്യൂറിങ് ദിസ് പ്രോസസ്സ്,അനെസ്തേസിയോളജിസ്റ് വിൽ ടേക്ക് കെയർ ട്ടോ പ്രൊട്ടക്ട യുവർ ടീത് ആൻഡ് മൗത് ഫ്രം ഇഞ്ചുറി .

വാട്ട് ഏറെ ദി റിസ്ക്സ് ആൻഡ് സൈഡ്-എഫക്ട് ?

വെ ഡോനോട് എക്സ്പെക്ട് ദാറ്റ് ഔർ സ്റ്റഡി വിൽ ക്സ് എനി ഇഞ്ചുറി ട്ടോ യു ബെചൌസ് ഔർ സ്റ്റഡി പ്രോട്ടോകോൾ ഈസ് പാർട്ട് ഓഫ് റൂട്ടിനെ ഇന്ത്രൊപ്പറേറ്റീവ് ട്രാൻസ് ഈസോഫഗെയ്ൽ ക്സാമിനേഷൻ . യു വിൽ ബി അണ്ടർ ദി എഫക്ട് ഓഫ് അനസ്തേഷ്യ വൈലെ ദി ടെസ്റ്റ് ഈസ് ബേക്കിങ് പെർഫോർഡ്,തുസ് യു ഏറെ ഉൺലിക്കലി ട്ടോ എക്സ്പീരിയൻസ് എനി ഡിസ്കഫോട് .

വൈ ഏറെ വെ ടോണിങ് ദിസ് സ്റ്റഡി ?

ദി പുർപ്പോസ് ഓഫ് ദിസ് സ്റ്റഡി ഈസ് ട്ടോ എസ്സ് ദി യൂസേഫുൽനെസ്സ് ഓഫ് ഗ്ലോബൽ ലോങ്ങിട്ടിനാൽ സ്ട്രെയിൻ ഫോർ പ്രെഡിക്റ്റിങ് പോസ്റ്റ്-ഓപ്പറേറ്റീവ് ഔറ്കോംസ് ഇൻ പടൈന്റ്സ് ഉണ്ടർഗോയിങ് ക്യാബിഗ് വിത്ത് പ്രൊപോഫോൾ ഓർ സെവോഫ്ലുരനെ അനസ്തേഷ്യ . ഇറ്റ് വിൽ ആൾസോ ഹെല്പ് ട്ടോ സ്പ്ലൊറേ ഫുർത്തെറ ഇത് യൂട്ടിലിറ്റി ഡ്യൂറിങ് കാർഡിയാക് സർജറി

ക്യാൻ യു വിതദരൌ ഫ്രം ദിസ് സ്റ്റഡി ആഫ്റ്റർ ഇത് സ്റ്റേറ്റ്സ് ?

യുവർ പാര്ടിസിപാഷൻ ഇൻ ദിസ് സ്റ്റഡി ഈസ് എന്ടിറേലി വോളണ്ടറി ആൻഡ് യു ഏറെ ആൾസോ ഫ്രീ ട്ടോ ഡിസൈഡ് ട്ടോ വിതദരൌ പെർമിസ്സിഷൻ ട്ടോ

പാർട്ടിസിപ്പറ്റി ഇൻ ദിസ് സ്റ്റഡി . ഇഫ് യു ദോ സൊ , ദിസ് വിൽ നോട എഫ്ഫെക്ട് യുവർ യൂസർൽ ട്രീത്തെമന്റ് അറ്റ് ദിസ് ഹോസ്പിറ്റൽ ഇൻ എനി വായ്

വാട്ട് വിൽ ഹാപ്പെൻ ഇഫ് യു ഡെവലപ്പ് എനി സ്റ്റഡി റിലേറ്റഡ് ഇഞ്ചുറി ?

വെ ഡോനോട് എക്ഷ്പെക്ട് എനി ഇഞ്ചുറി ട്ടോ ഹാപ്പെൻ ട്ടോ യു ബെചൌസ് ഓഫ് ഔർ സ്റ്റഡി ആസ് ഇറ്റ് ഈസ് എ പാർട്ട് ഓഫ് റൂട്ടിനെ ക്സാമിനേഷൻ ഡ്യൂറിങ് ദി സർജറി

വിൽ യു ഹാവ് ട്ടോ പേ ഫോർ ദി സ്റ്റഡി? നോ

വിൽ യുവർ പേർസണൽ ഡീറ്റെയിൽസ് ബി കേപ്ട് കോണ്ഫിഡന്റിൽ?

യുവർ പേർസണൽ ഡീറ്റെയിൽസ് വിൽ ബി കേപ്ട് കോണ്ഫിഡന്റിൽ. ദി റിസൾട്ട് ഓഫ് ദിസ് സ്റ്റഡി വിൽ ബി പുബ്ലിഷ്ഡ് ഇൻ എ മെഡിക്കൽ ജേർണൽ ബട്ട് യു വിൽ നോട ബി ഐഡന്റിഫൈഡ് ബൈ നെയിം ഇൻ എനി പുബ്ലിക്കേഷൻ ഓർ പ്രസന്റേഷൻ ഓഫ് റിസൾട്സ്

ഇഫ് യു ഹാവ് എനി ഫുർത്തെറ കുഎസ്ടിഒന്സ്, പ്ളീസ് ആസ്ക്,

Dr. Chennakeshavallu G N, Senior Resident, Department of Anaesthesia (Ph No: 9914207949)

Dr.Rupa Sreedhar, Professor and Head of Department of Anaesthesia.

Dr.Shrinivas Gadhinglajkar, Professor, Department of Anaesthesia.

Dr.Prasant kumar Dash, Professor, Department of Anaesthesia.

Dr.Prashant Bhaskar , Senior Resident, Department of Anaesthesia

ഡിക്ലറേഷൻ

ഐ, ----------(പാർട്ടിസിപ്പന്റ്'സ് നെയിം /അജ്)സോങ്/ഡൗഗ്റ്റർ ഓഫ് ----------

ഡിക്ലറേ ദാറ്റ് ഐ റെയ്ഡ് ദി എബോവ് ഇൻഫർമേഷൻ പ്രോവിടെ ട്ടോ മി റെഗാർഡിങ് ദി സ്റ്റഡി (പ്ളീസ് ടിക്ക് ബോസ്സ്).

ടൈറ്റിൽ ഓഫ് ദി സ്റ്റഡി: ഗ്ലോബൽ ലോങ്ങിട്ടിനാൽ സ്ട്രെയിൻ ഫോർ ഇവള്അഷൻ ഓഫ് പോസ്റ്റ് ഓപ്പറേറ്റീവ് ഔറ്കോമേ ഇൻ പടൈന്റ്സ് ഉണ്ടർഗോയിങ് കൊറോണറി ആർട്ടറി ബിപാസ്സ് ഗ്രാഫ്റ്റിങ് റേസിവിങ് ഈതെർ സെവോഫ്ലുരനെ ഓർ പ്രൊപോഫോൾ അനസ്തേഷ്യ

· ആൻഡ് ഹാവ് ക്ലാരിഫൈഡ് എനി ഡൗബ്റ്റ്സ് ദാറ്റ് ഐ ഹെഡ്.[ ]

· ഐ ആൾസോ ഉണ്ടെർസ്ടന്ദ് ദാറ്റ് മൈ പാർടിസിപാഷൻ ഇൻ ദിസ് സ്റ്റഡി ഈസ് എന്ടിറേലി വോളണ്ടറി ആൻഡ് ദാറ്റ് ഐ ആം ഫ്രീ ട്ടോ വിതദരൗ പെർമിസ്സിഷൻ ട്ടോ കന്റിണ്ൊ ട്ടോ പാർട്ടിസിപ്പറ്റി അറ്റ് എനി ടൈം വിതൗട് അഫക്റ്റിങ് മൈ യൂസുല് ട്രീതെമന്റ് ഓർ മൈ ലെങൾ റൈറ്സ്[ ]

· ഐ ഉണ്ടെർസ്ടന്ദ് ദാറ്റ് ദി സ്റ്റഡി സ്റ്റാഫ് ആൻഡ് ഇന്സ്ടിട്യൂഷ എത്തിക്സ് കമ്മിറ്റി മെംബേർസ് വിൽ നോട നീഡ് മൈ പെർമിസ്സിഷൻ ട്ടോ ലുക്ക് അറ്റ് മൈ ഹെൽത്ത് റെക്കോർഡ്സ് ഈവൻ ഇഫ് ഐ വിതദരൗ ഫ്രം ദി ട്രയൽ. ഐ അഗ്രി ട്ടോ ദിസ് അക്സസ്സ് .[ ]

· ഐ ഉണ്ടെർസ്ടന്ദ് ദാറ്റ് മൈ ഐഡന്റിറ്റി വിൽ നോട ബി റെവെൽഡ് ഇൻ എനി ഇൻഫർമേഷൻ റീലീസ്ഡ് ട്ടോ തേർഡ് പാർട്ടീസ് ഓർ പുബ്ലിഷ്ഡ് [ ]

· ഐ വോളന്ററിലി അഗ്രി ട്ടോ ടേക്ക് പാർട്ട് ഇൻ ദിസ് സ്റ്റഡി [ ]

· ഐ റേസിവ്ഡ് എ കോപ്പി ഓഫ് ദിസ് സൈനേഡ് കോൺസെന്റ് ഫോം [ ]

നെയിം: നെയിം ഓഫ് വിറ്റൻസ് :

സൈനർ: റിലേഷൻ ട്ടോ പാർട്ടിസിപ്പന്റ്:

തീയതി:

(പേഴ്സൺ ഒബ്ടൈനിംഗ് കോൺസെന്റ്)

ഐ അറ്റെസ്റ് ദാറ്റ് ദി റുകിർമെൻറ് ഫോർ ഇൻഫോംഡ് കോൺസെന്റ് ഫോർ ദി മെഡിക്കൽ റിസർച്ച് പ്രൊജക്റ്റ് ടെസ്ക്രൈബേഡ് ഇൻ ദിസ് ഫോം ഹാവ് ബീന സാറ്റിസ്ഫൈഡ് . ഐ ഹായ് ഡിസ്ക്കസ്ഡ് ദി റിസർച്ച് പ്രൊജക്റ്റ് വിത്ത് ദി പാർട്ടിസിപ്പന്റ് ആൻഡ് സ്പ്ളിനെക് ട്ടോ ഹിം ഓർ ഹേർ ഇൻ നോൺ ടെക്നിക്കൽ റ്റർ ഓൾ ഓഫ് ദി ഇൻഫർമേഷൻ കണ്ടൈൻഡ് ഇൻ ദിസ് ഇൻഫോംഡ് കോൺസെന്റ് ഫോം, ഇൻക്ലുഡിങ് എനി റിസ്ക്സ് ആൻഡ് അഡ്വെർസ് റീക്ഷൻസ് ദാറ്റ് മെയ് റീസണബിലി ബി സ്പെക്ടഡ് ട്ടോ ഓസിക്ക. ഐ ഫുർത്തെറ സെർടിഫിയ ദാറ്റ് ഐ എൻകോജ്ഡ് ദി പാർട്ടിസിപ്പന്റ് ട്ടോ ആസ്ക് കുഎസ്ടിഒന്സ് ആൻഡ് ദാറ്റ് ഓൾ കുഎസ്ടിഒന്സ് എസ്കേഡ് വെറെ അന്സെവർഡ്

Dr. Chennakeshavallu G N,

Senior Resident, CVTA,

SCTIMST

OBSERVATION CHART

TITLE: "Effect of sevoflurane versus propofol on segmental and Global longitudinal strain in patients undergoing coronary artery bypass surgery"

Observation chart

The following information will be recorded in both the groups

Pre-operative information

Name: Age: Weight:

Gender: Hospital number: Date:

Diagnosis and surgery:

Pre-operative medications:

Pre-operative TTE findings:

Coronary angiogram findings/ Left ventricular segmentation
:

Segment No.	Number of Normal segments	Number of ischemic/ Diseased(>70% stenosis)
LAD		
LCX		
RCA		

Co-morbidities:

Intra-operative data

1. Hemodynamics data:

Time	HR	IBP	CVP
Baseline			
30 min			
1hr			
1hr 30min			
2 hr			

2. Pre-CPB echocardiographic data:

Parameter	
End-systolic volume (ml)	
End-diastolic volume (ml)	

Ejection fraction(%)	
Stroke volume index (ml/m^2)	
Cardiac index (litre/min/m^2)	
Global longitudinal strain(%)	

3. Surgical data:

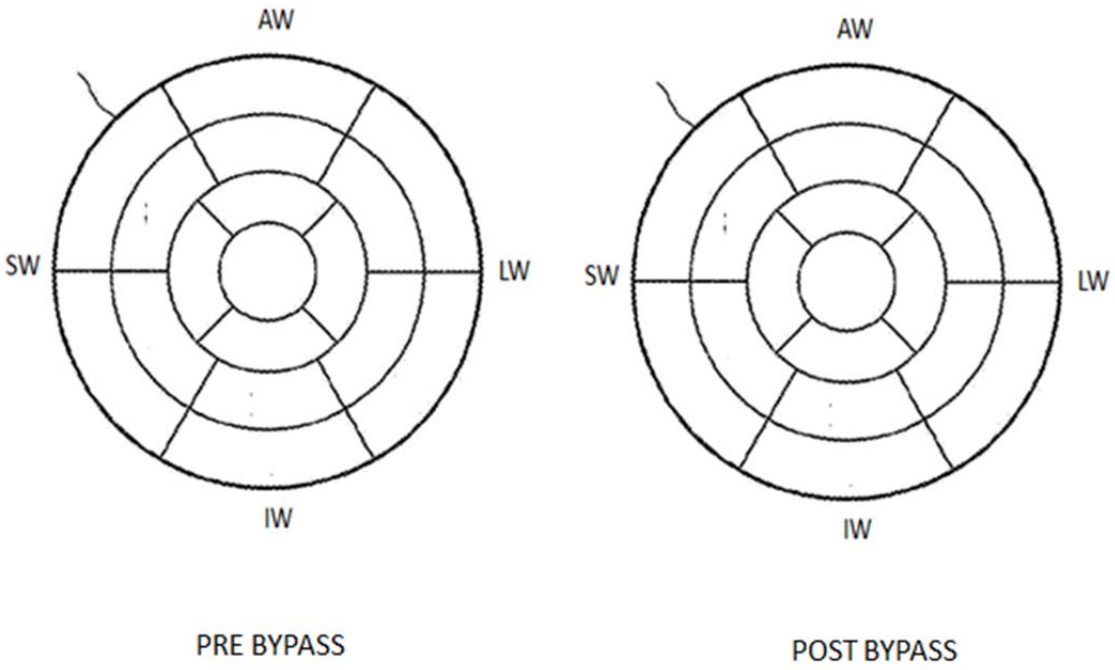
Parameter	
No. of bypass grafts	
No. of arterial grafts	
Aortic cross clamp time(min)	
CPB time(min)	
Pre-CPB time(min)	
Post-CPB time(min)	
Perfusion pressure	
CPB Flow	

4. Post-CPB echocardiographic data:

Parameter	
End-systolic volume (ml)	
End-diastolic volume (ml)	
Ejection fraction (%)	
Stroke volume (ml/m^2)	
Cardiac output (litre/min/m^2)	
Global longitudinal strain (%)	

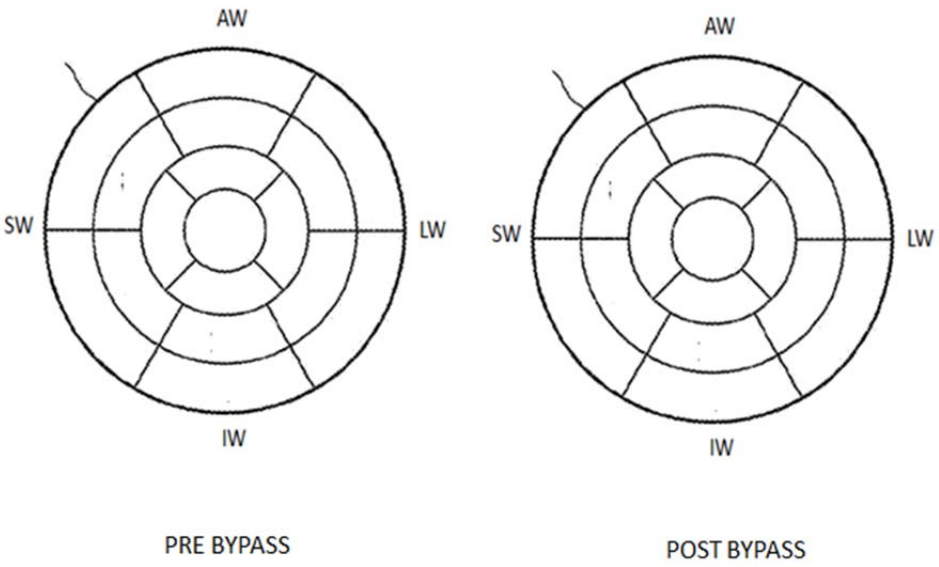
5. Wall motion score and segmental longitudinal strain in pre-CPB and post-CPB period will be represented in bull's eye format

Bull's eye representation of regional wall motion score.



Wall motion	Pre-bypass (No. of segments)	Post-bypass (No. of segments)
Normal		
Hypokinesia		
Akinesia		
Dyskinesia		
WMSI		

Bull's eye representation of Segmental strain.



Post-operative data

Parameter		
Inotrope 1	Inotrope 2	Inotrope 3
Dose:	Dose:	Dose:
Duration(min):	Duration(min)	Duration(min)
Inotropic score		
Duration of mechanical ventilation (hours)		
Duration of ICU stay (Hours)		

Inotropic score = 100 X Epinephrine (mcg/kg/min) +100 X Nor-epinephrine (mcg/kg/min)

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)
Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

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

SCT/IEC/1011/DECEMBER-2016 25.10.2017

Dr. Chennakeshavallu G. N
Senior Resident
Department of Anaesthesiology
SCTIMST, Thiruvananthapuram

Dear Dr. Chennakeshavallu,

The Institutional Ethics Committee reviewed and discussed your application to conduct the study entitled "GLOBAL LONGITUDINAL STRAIN FOR EVALUATION OF POST-OPERATIVE OUTCOME IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS GRAFTING RECEIVING EITHER SEVOFLURANE OR PROPOFOL ANESTHESIA" (IEC/1011) on 17th December, 2016.

The following documents were reviewed:

Original submission

1. Covering letter addressed to the Chairman, IEC, SCTIMST, dated 25.11.2016 with checklist
2. TAC Approval Letter
3. IEC Application Form
4. Project Proposal
5. Observation chart
6. Declaration Form
7. Consent Forms in English and Malayalam
8. CV of Principal Investigator and Co- Principal Investigators

Revised submission

1. Covering letter addressed to the Chairman, IEC, SCTIMST, dated 07.10.2017 with checklist
2. TAC Approval Letter
3. IEC Application Form
4. Project Proposal
5. Observation chart
6. Declaration Form
7. Consent Forms in English and Malayalam
8. CV of Principal Investigator and Co- Principal Investigators

The following members of the Ethics Committee were present at the meeting held on 17th December, 2016 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. Harikrishna Varma PR	PhD	Male	Biomedical Scientist	Yes
3.	Dr. Meenu Hariharan	DM	Female	Clinician (Gastro-Enterologist)	No
4.	Dr. Rema M. N	MD	Female	Pharmacologist	No
5.	Dr. R V G Menon	PhD	Male	Lay Person	No
6.	Smt. Sathi Nair	MA	Female	Lay Person	No
7.	Dr. K R S Krishnan	ME, PhD	Male	Biomedical Scientist/Engineer	No
8.	Dr. Kala Kesavan. P	MD	Female	Pharmacologist	No
9.	Dr. Christina George	MD	Female	Psychiatrist	No
10.	Dr. P. Manickam	PhD	Male	Scientist - Epidemiologist	No
11.	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

Plagiarism Checker X Originality Report

Similarity Found: 3%

Date: Monday, July 30, 2018
Statistics: 265 words Plagiarized / 7990 Total words
Remarks: Low Plagiarism Detected - Your Document needs Optional Improvement.

INTRODUCTION Left ventricular (LV) systolic function is a predictor for perioperative morbidity and mortality in patients undergoing coronary artery bypass grafting (CABG)1-3. Transesophageal echocardiography (TEE) is a corner-stone for assessing LV systolic function in routine intraoperative practice. Conventional methods for analysis of regional and global LV systolic function use qualitative/quantitative approach like visual assessment of inward radial motion and wall thickening, FS , FAC , SV, EF).

These methods have many limitations including necessity to make geometric assumptions, less reproducibility, inter-observer variability, load-dependence, influence of hemodynamic variables and translation motion4,5. Echocardiographically derived wall motion severity index has good ability to predict outcomes after CABG6. However, this method has limitations which include need for significant expertise, lack of precise distinction between varying degrees of hypokinesia and potential failure to identify subtle abnormalities.

7 Endocardial wall motions reflect only the radial contractile function of the heart neglecting the contribution of longitudinal myocardial deformation which is affected at initial stages of ischemia. Furthermore, impairment of regional wall motion score does not cause reduction in LVEF unless several segments are involved. Also, tethering of a normally contracting tissue to the adjacent infarcted area may also appear akinetic accounting for overestimation of infarct size by WMS8.Several studies suggest that LVEF is poorly sensitive in detecting early myocardial dysfunction9.

There is a growing body of evidence showing that the assessment of myocardial deformation by Doppler or speckle tracking techniques is a sensitive and robust index to detect subclinical myocardial dysfunction with defined normal range and

providesINTERNET SOURCES:

0% - http://atm.amegroups.com/article/view/29
0% - https://www.bing.com/aclick?ld=d3PCP26Ea
0% - https://link.springer.com/chapter/10.100
0% - Empty
0% - http://twri.tamu.edu/docs/bacteria-tmdl/
0% - http://onlinelibrary.wiley.com/doi/10.11
0% - http://www.onlinejacc.org/content/6/3/58
0% - https://link.springer.com/content/pdf/10
0% - https://www.researchgate.net/publication
0% - http://www.onlinejacc.org/content/42/3/5
0% - http://www.escoladeeco.com.br/esquina_ci
0% - https://academic.oup.com/ehjcimaging/art
0% - http://openheart.bmj.com/content/1/1/e00
0% - https://157030693.r.bat.bing.com/?ld=d3Z
0% - https://www.sciencedirect.com/science/ar
0% - https://academic.oup.com/ehjcimaging/art
0% - https://patents.google.com/patent/US5724
0% - https://www.researchgate.net/publication
0% - https://www.bing.com/aclick?ld=d39E4pv-C
0% - http://journals.sagepub.com/doi/pdf/10.1
0% - http://europepmc.org/articles/PMC4066464

Patient no.	GROUP	Hospital no.	WEIGHT	age	Sex	preoperative medicationS	pre operative TTE
1	S	427083	59	69	M	METOLAR ISOSORBIDE ATORVA	LV- 43/23, EF-78%, NO RWMA
2	S	425304	55	58	M	METOLAR ATORVA DIBIZIDE IMDUR	LV=44/29, EF=%, NO RWMA
3	S	473807	59	48	M	ATORVA METFORMIN METOLAR	LV-45/30, EF60%, NO RWMA
4	S	427569	72	52	M	ATORVA METOLAR	LV=45/28, EF=67% SEP 18/14/ GD LV NO RWMA
5	S	170208	66	60	M	ATORVA METPPROLOL GLIMEPRIDE SORBITRATE	LV EF = 79% NO RWMA
6	S	426759	52	55	M	METOLAR ROZAT	LV= 52/25, EF= 80%, NO RWMA
7	S	422606	63	77	F	ROSUVA, AMLONG	LV= 44/26, EF=72% NO RWMA
8	P	442321	53	57	M	ROZAT, METOLAR, GLIMISTAR NOVOMIX	LV= 58/40, EF=51% RWMA= AW/AS HYPOKINESIA, FAIR LV
9	P	442238	63	70	M	METOLAR, ROSUVA, SALBUTAMOL INHALER	LV=51/3, EF=55%, RWMA=IW BASAL/MID, LATERAL= BASAL/MID GOOD LV
10	P	434793	75	46	M	METOPROLOL AMLONG IMDUR SORBITRATE, ATORVA	LV 49/36, EF 54%, SEP 16/12, PW 14/12, CONC LVH, GRADE 1 dd , no rwma, good lv mild mr
11	P	423405	**68**	63	M	METFORMIN GLIMEPRIDE METOLAR ATORVA INSULIN	LV 45/30, EF-62% SEP 13/9, GD BIVENTRICULAR FUNCTION
12	P	440255	74	61	M	METFORMIN METOLAR ATORVA	GOOD BV FUNCTION NO RWMA NO PE, NO MR
13	P	445677	70	60	M	METFORMIN METOPROLOLO ATORVA	LV 46/26 EF 73% SEP 16/11, GD LV , CONC LVH, GRADE 1 DD,
14	P	440623	68	74	M	ROZAT METOLAR GLYCIPHAGE	LV= 54/38, EF= 50%, MILD LVD,
15	P	446507	74	65	M	METFORMIN METOLAR ATORVA INSULIN	LV 52/36, EF 58% GOOD LV , MILD MR
16	P	444016	77	72	M	METOPROLOL, METFORMIN. IMDUR ATORVA	LVID-47/20, EF=68%, Conc LVH, No RWMA, No MR/TR
17	S	443346	58	43	M	ATENOLOL INSULIN	LV=51/33 EF=68% NO RWMA, GOOD LV
18	P	441586	70	53	M	TENOLOL METOLAR	LV=53/3, EF=57%, NO RWMA TRIVIAL MR , CONC LVH, GOOD LV
19	S	426353	70	41	M	ROZAT DILZEM	LV=47/24 EF -65%, NO RWMA
20	S	256210	55	59	M	ROZAT SORBIDE METOLAR METFORMIN	LV=50/27, EF =58%, NO RWMA MILD MR
21	P	434266	68	73	F	METOLAR METOLAR	LV= 48/30, RWMA PRESENT IN BASAL MID AW AS IW
22	S	435504	74	48	M	METFORMIN METOLAR ATORVA	LV= 47/34, EF=54%, RWMA= MID/APICAL AS HYPOKINESIA
23	S	439404	70	47	F	METOLAR	LV=41/29, EF=60%, RWMA= BASAL IW GOOD LV
24	S	417608	62	70	M	METOLAR , NITROCORTIN IMDUR, SALBUTAMOL	LV= 46/32, EF=58%, NO RWMA
25	S	438374	57	59	F	GLICAZIDE, INSULIN, METOLAR CARDACE RANOLAZINE ROSUVA	LV=61/51, EF 45% MILD MR , RWMA= AW/AL/AS HYPOKINETIC
26	P	574709	55	71	M	ATORVA METTFORMIN GLICAZIDE, AMLODIPINE	LV-48/33, EF-55%, NO RWMA
27	P	437152	57	78	M	METOPROLOL AMLONG IMDUR	LV 48/30, EF - 4% SEP 17/13, RWMA , APICAL/MID/BASAL SEPTUM AW/AS, GOOD LV
28	S	438004	68	61	M	LASIX METOLAR, ROSUVA, TRIMETAZIDINE	LV 53/41, EF=54%, MILD LVD, RWMA= BASAL/MID AW/AS/AL,HYPOKINESIA , APEX HYPOKINESIA
29	S	425158	65	65	M	METOLOR, ATORVA	LVID 45/30, EF=60%, NO RWMA, MILD MR
30	P	443750	70	67	M	METOLAR ATORVA RANOLZINE, RAMPRIL, INSULIN	LVID 43/23, EF = 0%, NO RWMA, MR TRIVIAL
31	P	442910	74	51	M	THROXINE, ATORVA , RAMIPRIL	LV= 49%/34, EF=64%, NO RWMA, GOOD LV
32	P	434470	68	62	M	ROSUVA METOLAR RAMIPRUIL	LV = 53/47, EF 48%, MILD MR RWMA= AW/AS/AL BASAL/MID HYPOKINETIC, IW/IL HYPOKINESIA
33	P	441507	77	67	M	METFORMIN INSULIN, ATORVA, TRIMETAZIDINE, AMLONG	LV= 43/ 20, EF= 57%, NO RWMA , GOOD LV
34	S	436071	57	68	F	GLIMEPRIDE, METFORMIN, ROSUVA, METOPROLOL	LV= 48/ 38, EF=53%, RWMA = BASAL/MID IW/IL, LATERAL W ALL HYPOKINESIA
35	P	442239	61	58	M	METOLAR, ROZAT NOVOMIX METFORMIN	LV=49/23, EF= 58%, NO RWMA
36	S	285619	59	51	M	METOLAR, ROZAT	LV= 46/23, EF=60%, RWMA= IW/IS
37	P	444005	50	59	M	METFORMIN NOVOMIX ROZAT METOLAR	LV=56/43, EF=54%RWMA= AW/AS/AL HYPOKINETIC
38	S	213182	59	78	M	METOLAR	LV=52/24, EF= 80% NO RWMA
39	P	440054	70	52	M	NOVOMIX IDICIN, SELOKEN	LV= 39/25, EF=5%, NO RWMA
40	S	441037	68	52	M	GLYCIPHAGE, GALVAS, INSULOTARD	LV= 49/36, EF=55%, RWMA= BASAL MID IS/IW HYPOKINESIA

comorbiditeS	HR baSeline	HR 30 min	HR 1 HR	HR 1.5 MIN	HR 2 HR	HR 2.5 HR	HR POST CPB	MAP baSeline	MAP 30 min	MAP 1 MAP	MAP 1.5 MIN	MAP 2 MAP	MAP 2.5 MAP	MAP POST CPB	NTG USE	PE USE
COPD	50	69	70	77	70	65	69	110	89	89	100	89	89	89	NO	YES
DM	68	60	57	58	65	70	77	84	89	70	88	78	94	98	NO	YES
DM, HTN, DLP	77	80	70	68	64	68	70	70	77	68	89	70	93	80	YES	NO
HTN	48	57	60	64	63	69	72	98	99	92	110	90	92	98	YES	NO
DM DLP HTN SMOKER	70	87	88	80	82	78	77	100	110	95	89	78	87	98	NO	YES
DM HTN DLP	66	70	74	72	77	75	79	67	78	78	78	87	90	87	NO	YES
DLP	65	73	75	68	69	68	67	78	77	78	75	85	73	88	NO	NO
DM	70	74	75	77	70	72	73	94	98	88	77	84	72	99	NO	YES
ASTHMA, SMOKER	58	70	70	67	66	70	77	94	90	89	79	83	70	73	NO	YES
HTN CKD	65	70	77	68	77	80	89	110	84	77	80	80	78	74	NO	YES
DM	70	60	65	70	73	80	85	73	70	72	74	72	60	66	NO	YES
DM	79	70	80	88	80	84	70	86	98	90	89	89	89	98	NO	YES
DM DLP	53	55	65	70	58	60	78	97	83	80	77	78	78	74	NO	NO
DM, HTN, DLP	55	55	60	80	81	85	88	87	77	93	95	78	72	90	NO	NO
DM DLP	69	77	79	83	84	90	90	84	80	78	88	90	92	80	NO	NO
HTN, DM	70	80	89	78	90	90	98	89	80	77	75	75	70	65	NO	YES
DM DLP	50	67	77	60	71	68	80	80	98	89	87	80	80	78	YES	NO
DM HTN DLP	48	55	64	70	70	65	82	100	100	95	89	74	78	78	NO	NO
HTN	56	67	70	55	90	78	72	95	99	87	78	80	89	89	NO	NO
DM , HTN	70	77	68	55	60	70	70	80	78	99	77	88	90	90	YES	NO
	78	80	60	80	77	67	85	68	88	80	72	75	90	78	NO	YES
DM HTN	65	77	70	67	66	70	75	87	98	80	79	110	98	90	NO	YES
	60	90	77	55	55	70	77	98	95	84	77	80	99	70	NO	NO
DM, COPD, POVD	58	54	66	66	57	77	78	99	79	77	72	82	72	74	YES	NO
DM HTN	55	56	55	68	77	78	90	97	90	89	70	80	69	87	no	NO
DM HTN	54	53	57	58	80	76	88	88	88	99	70	77	80	90	NO	YES
HTN DLP	80	76	77	80	89	90	93	110	90	94	99	93	90	80	NO	YES
DLP, CKD	76	78	80	72	70	77	87	82	80	95	88	72	78	67	YES	NO
HTN	56	64	68	70	60	78	88	85	75	72	80	80	84	83	NO	NO
DM	64	60	58	59	70	58	80	97	88	79	75	75	68	97	NO	NO
HYPOTHYROIDISM	70	66	72	78	69	59	68	110	98	89	78	78	74	72	NO	NO
HTN	55	54	49	59	70	70	67	98	69	89	83	82	78	88	NO	YES
DM HTN DLP	70	77	58	55	70	68	79	77	94	90	98	74	80	89	NO	NO
DM HTN	59	45	49	55	55	60	70	84	89	84	80	78	85	98	NO	YES
DM HTN	55	54	49	59	70	77	67	80	78	90	87	78	77	77	NO	YES
HTN	53	70	68	77	58	78	89	77	94	90	98	74	80	89	NO	NO
DM HTN SMOKER	78	78	80	82	78	77	90	82	80	95	88	72	78	67	NO	NO
HTN	53	56	58	68	77	78	82	70	90	84	80	80	98	92	NO	YES
DM HTN DLP	80	76	77	80	89	90	93	80	78	99	77	88	90	90	NO	NO
DM HTN SMOKER, ULCERATIVE COLITIS	55	54	55	59	70	77	67	77	89	84	80	78	85	98	NO	YES

Patient no.	GROUP	Hospital no.	WEIGHT	age	Sex	preoperative medicationS	pre operative TTE
41	P	442278	58	54	M	INSULOTARD, LASIX	LV= 49/31, EF=67% NO RWMA, GOOD LV
42	S	374635	50	61	F	SELOKEN, ROZAT, LASIX, TRIBET	LV= 43/24, EF=75%, no rwma
43	S	449808	59	57	M	ROZAT, LASIX, SELOKEN SR GLYCOMET	LV=45/30, EF=64%, no rwma
44	S	449987	71	63	F	METOLAR, ATORVA, METFORMIN THYRONORM SORBITRATE	LV=52/37, EF=55% NO RWMA
45	P	449145	76	59	M	AZTOR, FLAVEDION, SORBITATE NITROCORTIN	LV=48/29, EF=70% NO RWMA
46	S	181521	60	71	M	METOPROLOL AZTOR CILACAR NITROCORTIN MIXATRD	LV-42/25, EF=60%, RWMA= MID IS/IW HYPOKINESIS
47	S	451463	49	60	F	METOPROLOL AMLODIPINE ATORVA ISOSORBIDE	LV= 55/40, EF=55%, RWMA= BASAL MID IW/IS HYPOKINESIS
48	S	446005	48	68	M	ATORVA, METOLAR, GLIMEPRIDE MIXTARD	LV=42/29, EF=59%, NO RWMA
49	P	446356	65	60	F	ATORVA ALDACTONE METOLAR LASIX INSULIN THYRONORM	LV=56/42, EF=53%, RWMA = AW/AS BASAL HYPOKINETIC
50	P	445442	68	43	M	METOLAR, AZTOR, GTN	LV=43/30, EF=56%, RWMA= IW/IS

comorbidites	HR baSeline	HR 30 min	HR 1 HR	HR 1.5 MIN	HR 2 HR	HR 2.5 HR	HR POST CPB	MAP baSeline	MAP 30 min	MAP 1 MAP	MAP 1.5 MIN	MAP 2 MAP	MAP 2.5 MAP	MAP POST CPB	NTG USE	PE USE
DM, CKD	64	55	54	59	77	58	80	84	88	79	75	75	68	97	NO	YES
DM, DLP	53	56	58	68	77	78	82	80	78	90	87	78	77	77	NO	YES
dm htn smoker	70	77	58	55	70	68	79	110	98	89	78	78	74	72	NO	YES
DM HTN HYPOTHYROID	78	78	80	88	79	68	90	98	69	89	83	82	78	88	YES	NO
HTN SMOKER	78	78	80	88	79	68	90	85	75	72	80	80	84	83	no	NO
DM	55	54	49	59	70	70	67	77	94	90	98	74	80	89	NO	YES
HTN	78	78	80	88	79	68	90	84	89	84	80	78	85	98	NO	YES
DM, HTN, DLP	78	78	80	88	79	68	90	85	75	72	80	80	84	83	no	NO
DM,HTN	77	72	68	66	80	71	72	89	92	78	82	80	79	84	NO	YES
NIL	65	70	76	80	70	67	80	94	98	89	80	93	90	92	NO	YES

CORONARY ANGIOGRAM

Patient no.	REPORT	BA	BAS	BIS	BI	BIL	BAL	MA	MAS	MIS	MI	MIL	MAL	AA	AS	AI	AL	A	NUMBER OF NORMAL SEGMENTS	NUMBER OF HYPOPERFUSED SEGMI
1	LMCA- N, LAD-3, OSTIOPROX 80%, LCX- 80% , RCA- D, MILD PLAQUES	I	I	N	N	I	I	I	I	N	N	I	I	I	I	N	I	I	5	12
2	LMCA- N, LAD- DIFFUSE DISEASE, PROX LAD 80%, MID LAD- DIFFUSE, LCX- ND OP- 0-70%, RCA-D, OP TIGHT LESION	I	I	I	I	N	N	I	I	I	I	N	N	I	I	I	N	I	5	12
3	LMCA-N, LAD-TYPE 3 PROX 80%, LCX 70%, RCA-90%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
4	LMCA=N, LAD=TYPE1 LOND SEG SUBTOTAL OCCLUSION, LCX= 60%, RCA=D, 80% DISEASES	I	I	I	I	N	N	I	I	I	I	N	N	I	I	I	N	I	5	12
5	LMCA =N, LAD=TYPE3 MID TOTAL OCCLUSION , RCA= OSTIAL TIGHT	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
6	LMCA= N, LAD= 3, PROX CTO, LCX= D, PROX 80%, RCA= D, CTO MID	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
7	LMCA= N, LAD= TYPE 3 PROX 70-80% CALCIFIC, MID =70%, LCX = ND, OM =708%, RCA= ANAMOLOUS ORIGIN FROM LCC, MID =LONG DIFFUSE 80-90%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
8	LMCA= N, LAD= OSTIAL 80% MID 90%, LCX= ND, OSTIAL 50% DISTAL 70% RCA =D, PROX 90%	I	I	I	I	N	N	I	I	I	I	N	N	I	I	I	N	I	5	12
9	LMCA= N, LAD = TYPE 3 , PROX CTO, DIAGONAL STENTED , ISR, LCX=ND, PROX TOTAL ISR, RCA =D, NO FLOW LIMITATION	I	I	N	N	I	I	I	I	N	N	I	I	I	I	N	I	I	12	5
10	LMCA= N, LAD= 3, PROX 70%, MID 90%, LCX- ND, PROX 70%, RCA- D, PROX 30% DISTAL 70%	I	I	N	N	N	N	I	I	N	N	N	N	I	I	N	N	I	7	12
11	LMCA= DISTAL PLAQUE, LAD= TYPE3, PROXIMAL 70%, DISTAL 70%, LCX= 80% DISEASE, RCA DOMINANT PROXMIAL 30%DISTAL NEAR TOTAL	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
12	LMCA = N, LAD TYPE 3 PROXIMAL 80%, LCX=80%, RCA= CTO PROX	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
13	LMCA =N, LAD=DIFFUSELY DISEASED, LCX=90 STENOSIS, RCA= DOMINANT , MID SEG 99%DISEASED	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
14	LMCA= 50% DISEASE, LAD DISEASED FROM OSTIUM, TOTAL OCCLUSION PROXIMALLY, CALCIFIED, LCX= 70% RCA= DOMINANT, PROX 90% DISEASED	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
15	LMCA=N, LAD= TYPE 3, PROX 80%, LCX =ND, PROX 90%, RCA=D, MID TOTAL	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
16	LMCA=distal 50-60%, LAD=prox 50-60%,LCX=mild ostial disease, RCA =prox total, dominant	N	N	I	I	N	N	N	N	I	I	N	N	N	N	I	N	N	5	12
17	LMCA=N, LAD= TYPE 3 PROX 60%, LCX =TOTAL OCCLSION, RCA= MID 60% DISEASED	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
18	LMCA= N, LAD= TYPE 3 PROX TOTAL, LCX= PROX 80%RCA = 30%	I	I	N	N	I	I	I	I	N	N	I	I	I	I	N	I	I	5	12
19	LMCA-N , LAD3, DISTAL DIFFUSE DISEASE , LCX -ND OSTIOPROX 80%, RCA - DIFFUSELY DISEASES	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
20	LMCA-SHORT, LAD-TYE 3, PROX 70%, DISTAL DIFFUSE DISESES, RCA -D, CTO, LCX PROX 80%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
21	LMCA= DIATAL 70%, LAD= TYPE3 OSTIAL=60%, PRO=70%, LCX= ND, OSTIAL 80%, RCA=D, PROX 80%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
22	LMCA=N, LAD = DISTAL ISR99%, LCX= DISTAL 90%, RCA=LONG SEGMENT TOTAL OCCLUSION	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
23	LMCA= N, LAD= PROX 70%, MID =80-90%, LCX- ND, MID 60%, RCA-D, MID 50-60%	I	I	N	N	N	N	I	I	N	N	N	N	I	I	N	N	I	10	7
24	LMCA= N, LAD= 3, PROX MID CTO, LCX- ND, PROX 70%, RCA= PROX TOTAL , DOMINANT	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
25	LMCA= N, LAD= TYPE3 PROX= 99%, LCX= PROX NEAR TOTAL, RCA=MID TOTAL	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
26	LMCA-N. LAD- TYPE 3, OSTIOPROX 50%, DISTAL 80%, RCA, D 80%, LCX-ND PROX 50% DISTAL 70%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
27	LMCA=N, LAD=TYPE 3, CRITICAL 95%, PROX, ISR 50% RCA= MINOR ISR, LCX= 70% PROX	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
28	LMCA= NORMAL, LAD = TOTAL , LCX =90%, RCA=PROX 90%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
29	LMCA=N, LAD=MID CTO , RCA= PRECRUS 70%, LCX=60%	I	I	I	I	N	N	I	I	I	I	N	N	I	I	I	N	I		
30	LMCA= N, LAD=TYPE 3, MID LAD 80% , LCX =N, OM = MID CUT OFF,RCA = 30%, PDA=90%, PLB=90%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
31	LMCA= N, LAD= PROX 90%, LCX - BIFURCATION DISEASE, RCA= MID 50%	I	I	N	N	I	I	I	I	N	N	I	I	I	I	N	I	I	5	12
32	LMCA=N, LAD= TYPE 3 MID 0-70%, D1=70%, LCX= ND, 100% PROX ISR, RCA = TOTAL OCCLUSION PROX	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
33	LMCA= 50% OSTEOPROXIMAL, LAD TYPE3,60-70% DIFFUSE , DIAGONAL= PROX 80%, LCX= ND, NORMAL, RCA = D, 50% PROX	I	I	N	N	N	N	I	I	N	N	N	N	I	I	N	N	I	10	7
34	LMCA= N, LAD= PROX 60%, MID 85%, LCX PROX 70%, RCA DIFFUSE MID 90%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
35	LMCA= N, LAD= 3, PROX 80%, MID 90%, LCX=ND DISTAL 70%, RCA=D, PROX 80-90%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
36	LMCA=N, LAD= 3, TOTAL AT D2, LCX= ND 50-60%, RCA- D, MID 90%	I	I	I	I	N	N	I	I	I	I	N	N	I	I	I	N	I	5	12
37	LMCA= N, LAD= PROX TOTAL LCX= ND, PROX 70%, RCA= D, 80%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
38	LMCA=N, LAD= 3, PROX OP 80% LCX= ND, MAJOR DISEASE, RCA= D, 80%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
39	LMCA=N, LAD= ECCENTRIC 90% DIFFUSE, MID PART, LCX= ND, TUBULAR ECCENTRIC 90%, RCA= D, TUBULAR 50%	I	I	N	N	I	I	I	I	N	N	I	I	I	I	N	I	I	5	12
40	LMCA=N, LAD= TYPE 3, PROX LOND SEG 99%, LCX= ND, MAJOR OM 40%, RCA= D, PROX TOTAL	I	I	I	I	N	N	I	I	I	I	N	N	I	I	N	I	I	5	12
41	LMCA= 30%, LAD= PROX 80%, DISTAL LAD= DIFFUSE , LCX= ND, OP 80%, DISTAL 70%, RCA= D, PDA=70%, PLB=70%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
42	LMCA=N, LAD= TYPE3, OP=70%, LCX=ND, DISTAL 70%, RCA= MID RCA 70%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
43	LMCA=DISTAL 60%, LAD= TYPE 3 , OP 40%, MID 90%, LCX=ND, MID TOTAL , RCA=D, MID 70% RAMUS =70%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
44	LMCA= 70%, LAD= 50%, RCA= D MID 80%, LCX= 80% PROX	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
45	LMCA=N, LAD= 3, PROX LAD TOTAL OCCLUSION, LCX= ND PROX 60%, RCA = TOTAL	I	I	I	I	N	N	I	I	I	I	N	N	I	I	N	I	I	5	12
46	LMCA= MID 40%, LAD= TOTAL OCCULDED , LCX= ND MID 90% RCA= D, NEAR TOTAL	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
47	LMCA= N, LAD= TYPE 3, OSTEOPROX CTO, LCX= ND, MID -70%, RCA- PROX MID DIFFUSE MID CTO	I	I	I	I	N	N	I	I	I	I	N	N	I	I	N	I	I	5	12
48	LMCA=N, LAD= TYPE 3 MID LAD TOTAL, LCX= ND, 50-60% RCA=D, 70%	I	I	I	I	N	N	I	I	I	I	N	N	I	I	I	N	I	5	12
49	LMCA=N,LAD= 90% MID, LCX= ECCENTRIC 70%, RCA= PROX 70-80%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17
50	LMCA= minor irregularities, LAD= TYPE3, PROX 60-70%, LCX= ND, MID 70%, OM=80%, RCA= D, PROX 80%	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	I	0	17

PRE OPERATIVE WMSI

Patient no.	BA	BAS	BIS	BI	BIL	BAL	MA	MAS	MIS	MI	MIL	MAL	AA	AS	AI	AL	A	GLOBAL WMSI
1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
4	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.52
5	1	2	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1
6	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.11
7	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
8	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1	1
9	1	1	1	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1.23
10	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1.23
11	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.05
12	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
13	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
14	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
15	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
16	1	1	1	2	1	1	1	1	1	2	1	1	1	1	1	1	1	1
17	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
18	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.11
19	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
20	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
21	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
22	1	1	1	1	1	1	1	2	1	1	1	1	1	2	1	1	1	1
23	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
24	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
25	2	2	2	2	2	2	2	2	2	1	1	1	2	2	1	1	1	1
26	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.11
27	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1
28	1	1	1	1	1	1	2	2	1	1	1	1	2	1	1	1	1	1
29	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.64
30	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
31	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.05
32	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1	1.17
33	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
34	1	1	1	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1
35	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
36	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
37	1	2	1	2	1	1	1	2	1	2	1	1	1	1	1	1	1	1.23
38	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
39	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.23
40	1	1	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1
41	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
42	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.23
43	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
44	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
45	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.23
46	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
47	1	1	1	2	2	1	2	1	1	2	2	1	1	1	1	1	1	1
48	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
49	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1	1
50	1	1	1	2	1	1	1	1	1	2	1	1	1	1	1	1	1	1

PRE OPERATIVE STRAIN QUANTIFICATION

Patient no.	GROUP	BA	BAS	BIS	BI	BIL	BAL	MA	MAS	MIS	MI	MIL	MAL	AA	AS	AI	AL	A	A4C	A2C	A3C	GLS
1	S	9-	6-	17-	10-	11-	11-	13-	28-	17-	20-	6-	11-	23-	21-	18-	18-	20-	12-	15-	15-	17-
2	S	8-	12-	8-	12-	18-	13-	12-	16-	11-	13-	14-	21-	18-	12-	14-	15-	14-	11-	9-	13-	11-
3	S	19-	12-	11-	23-	26-	11-	20-	15-	15-	19-	14-	21-	22-	19-	20-	18-	19-	16-	20-	17-	18-
4	S	12-	12-	10-	17-	16-	21-	11-	10-	12-	20-	19-	11-	16-	16-	18-	13-	15-	16-	13-	11-	13-
5	S	11-	7-	13-	14-	15-	15-	13-	27-	11-	20-	16-	16-	17-	15-	10-	8-	6-	14-	10-	12-	10-
6	S	12-	11-	7-	14-	9-	14-	13-	8-	18-	17-	4-	13-	17-	25-	9-	11-	3-	13-	15-	8-	9-
7	S	7-	9-	12-	10-	17-	13-	10-	24-	21-	N	17-	22-	21-	22-	11-	16-	18-	17-	N	16-	16-
8	P	16-	13-	13-	6-	11-	13-	17-	8-	12-	7-	10-	12-	7-	10-	17-	3-	4-	6-	10-	7-	13-
9	P	14-	21-	16-	4-	7-	12-	13-	21-	13-	17-	14-	10-	7-	10-	15-	5-	6-	7-	12-	9-	13-
10	P	9-	13-	15-	15-	12-	18-	6-	5-	17-	11-	17-	5-	14-	15-	20-	15-	15-	10-	11-	11-	14-
11	P	10-	10-	18-	12-	18-	13-	13-	10-	19-	21-	6-	8-	21-	22-	14-	17-	22-	17-	12-	20-	11-
12	P	11-	14-	14-	5	17-	12-	20-	14-	16-	14-	20-	8-	23-	19-	21-	15-	20-	9-	16-	14-	15-
13	P	10-	8-	14-	20-	11-	14-	18-	13-	13-	14-	13-	12-	16-	18-	14-	12-	19-	17-	20-	10-	17-
14	P	6-	14-	13-	9-	13-	14-	6-	12-	15-	11-	16-	16-	7-	19-	18-	7-	11-	13-	9-	9-	17-
15	P	15-	7-	4-	11-	22-	11-	21-	22-	17-	18-	18-	7-	12-	11-	15-	22-	25-	16-	20-	17-	11-
16	P	11-	15-	14-	16-	7-	21-	13-	13-	19-	18-	18-	20-	27-	23-	32-	23-	20-	20-	19-	18-	15-
17	S	21-	16-	16-	15-	15-	16-	17-	16-	17-	22-	12-	23-	17-	23-	15-	12-	18-	18-	16-	16-	14-
18	P	13-	13-	18-	7-	28-	13-	16-	21-	4-	15-	17-	12-	22-	29-	18-	21-	22-	17-	15-	16-	10-
19	S	6-	11-	14-	10-	17-	7-	16-	23-	13-	15-	17-	24-	21-	27-	17-	14-	19-	16-	14-	14-	9-
20	S	8-	6-	11-	15-	17-	11-	18-	20-	14-	24-	15-	10-	19-	21-	15-	20-	18-	21-	14-	19-	18-
21	P	17-	5-	9-	18-	18-	11-	12-	10-	14-	20-	16-	12-	14-	14-	20-	19-	16-	14-	15-	15-	14-
22	S	11-	6-	16-	7-	5-	13-	22-	11-	12-	9-	17-	8-	14-	15-	13-	19-	18-	12-	11-	16-	11-
23	S	4-	6-	13-	14-	13-	16-	8-	14-	13-	11-	15-	16-	14-	22-	16-	21-	18-	17-	9-	13-	18-
24	S	10-	13-	11-	10-	17-	11-	15-	17-	15-	12-	8-	8-	12-	11-	17-	10-	15-	22-	13-	9-	16-
25	S	13-	14-	11-	11-	9-	3-	4-	10-	6-	13-	8-	9-	9-	7-	10-	3-	4-	8-	8-	**6-**	10-
26	P	6-	8-	N	13-	17-	9-	14-	13-	10-	16-	13-	N	15-	8-	-12	8-	11-	7-	10-	12-	9-
27	P	6-	13-	16-	14-	18-	18-	19-	14-	13-	13-	11-	14-	20-	14-	23-	21-	20-	15-	17-	16-	12-
28	S	10-	13-	14-	9-	7-	3-	14-	18-	11-	12-	12-	9-	10-	8-	18-	17-	14-	10-	13-	11-	16-
29	S	13-	5-	3-	11-	20-	9-	8-	19-	15-	29-	10-	17-	16-	12-	13-	17-	24-	14-	15-	17-	8-
30	P	13-	13-	11-	5-	17-	12-	14-	11-	13-	12-	10-	11-	12-	21-	21-	13-	18-	13-	15-	12-	**9-**
31	P	13-	11-	7-	13-	12-	7-	14-	15-	17-	22-	18-	11-	23-	20-	19-	21-	21-	16-	15-	10-	10-
32	P	13-	3-	4-	5-	12-	9-	7-	6-	9-	8-	11-	4-	4-	15-	6-	8-	4-	7-	4-	3-	16-
33	P	13-	8-	11-	13-	17-	13-	20-	16-	21-	11-	22-	7-	24-	24-	17-	11-	28-	16-	20-	17-	13-
34	S	13-	5-	11-	10-	11-	6-	12-	17-	17-	8-	23-	22-	19-	11-	18-	15-	16-	13-	12-	15-	13-
35	P	13-	7-	13-	23-	18-	12-	13-	20-	11-	17-	6-	6-	14-	12-	14-	15-	14-	10-	15-	12-	11-
36	S	13-	3-	14-	9-	5-	12-	15-	14-	8-	12-	9-	18-	14-	18-	17-	12-	14-	13-	11-	4-	18-
37	P	13-	3-	10-	14-	7-	7-	7-	6-	13-	7-	6-	8-	12-	8-	13-	7-	8-	4-	11-	9-	19-
38	S	13-	9-	21-	16-	8-	15-	15-	16-	13-	16-	16-	18-	8-	19-	19-	16-	9-	22-	23-	8-	16-
39	P	13-	15-	3-	12-	12-	13-	15-	15-	17-	7-	10-	9-	15-	6-	19-	4-	10-	6-	10-	12-	14-
40	S	13-	23-	12-	11-	17-	13-	13-	17-	17-	19-	22-	19-	13-	12-	18-	11-	20-	23-	14-	18-	17-
41	P	13-	5-	16-	9-	20-	20-	19-	25-	13-	19-	16-	12-	10-	20-	14-	19-	20-	16-	15-	13-	15-
42	S	13-	8-	4-	5-	5-	14-	6-	10-	14-	16-	16-	14-	24-	21-	24-	18-	21-	14-	14-	15-	17-
43	S	13-	12-	16-	4-	9-	7-	3-	15-	9-	7-	13-	14-	14-	15-	5-	11-	13-	14-	10-	15-	13-
44	S	13-	13-	14-	13-	12-	21-	21-	19-	21-	19-	11-	16-	15-	22-	21-	20-	18-	19-	18-	19-	14-
45	P	13-	6-	11-	8-	13-	17-	8-	8-	6-	8-	6-	8-	11-	17-	23-	22-	21-	17-	14-	18-	7-
46	S	13-	13-	16-	18-	11-	21-	10-	12-	17-	25-	11-	13-	18-	14-	21-	17-	16-	16-	24-	14-	18-
47	S	13-	11-	12-	10-	15-	12-	13-	7-	11-	14-	11-	10-	13-	5-	11-	6-	9-	7-	11-	11-	11-
48	S	13-	7-	8-	4-	11-	9-	7-	10-	13-	17-	17-	18-	8-	11-	13-	17-	9-	9-	7-	9-	14-
49	P	13-	10-	8-	13-	12-	16-	7-	8-	9-	15-	12-	**16-**	4-	14-	17-	12-	15-	13-	11-	10-	11-
50	P	13-	13-	7-	9-	22-	14-	17-	19-	6-	3-	12-	9-	19-	20-	20-	20-	24-	11-	13-	25-	16-

PRE OPERATIVE 2D ECHO PARAMETERS					
patient no.	ESV(ml)	EDV(ml)	SVI(ml /m2)	CI(ml/m2)	EF(%)
1	25	68	43	2.5	63%
2	32	80	48	3.5	60%
3	34	88	54	3.8	61%
4	19	62	46	2.2	69%
5	55	120	56	3.5	70%
6	30	74	46	4	62%
7	14	59	45	3	48%
8	28	99	71	5.4	71%
9	23	78	55	4.4	70%
10	19	65	46	3.3	70%
11	33	80	47	2.6	58%
12	32	76	44	2.9	57%
13	24	79	55	3.1	69%
14	22	67	45	2.4	50%
15	29	69	40	3	52%
16	27	69	47	2.9	58%
17	25	65	40	3.2	61%
18	27	80	53	2.9	66%
19	24	68	44	3.4	64%
20	25	57	37	2.3	56%
21	25	56	31	1.9	55%
22	19	47	28	3.5	60%
23	17	52	36	3.4	67%
24	24	88	64	2.6	75%
25	42	94	22	2.6	55%
26	38	85	47	3.2	55%
27	39	82	43	2.6	66%
28	41	76	35	4.4	59%
29	28	68	40	2.8	58%
30	30	78	48	3.8	61%
31	24	49	25	1.9	51%
32	28	93	65	3.8	56%
33	32	68	36	2.4	52%
34	21	76	55	3	72%
35	26	46	24	1.8	52%
36	37	76	39	2.9	53%
37	23	60	37	2.5	61%
38	50	105	55	2.3	54%
39	24	57	33	2.4	66%
40	23	67	44	3.2	64%
41	19	61	42	3.4	65%
42	23	67	44	3.2	64%
43	19	46	27	3	59%
44	22	48	26	2	55%
45	23	85	62	4.2	72%
46	30	90	60	3.2	66%
47	24	58	36	2.1	58%
48	18	52	34	2.1	53%
49	25	69	44	3	53%
50	19	78	59	4.1	55%

POST OPERATIVE WMSI

Patient no.	Group	BA	BAS	BIS	BI	BIL	BAL	MA	MAS	MIS	MI	MIL	MAL	AA	AS	AI	AL	A	GLOBAL WMS
1	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
2	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
4	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
5	S	1	2	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1
6	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.11
7	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
8	P	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1	1
9	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.23
10	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
11	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
12	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
13	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
14	P	1	2	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1
15	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
16	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.11
17	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
18	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
19	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
20	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
21	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
22	S	1	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1
23	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
24	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
25	S	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1
26	P	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1	1.05
27	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
28	S	2	2	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1	1
29	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
30	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.23
31	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
32	P	1	2	2	1	1	1	1	2	2	1	1	1	1	2	1	1	1	1.23
33	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
34	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
35	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
36	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
37	P	2	2	2	1	1	1	2	2	2	1	1	1	2	2	1	1	1	1.29
38	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
39	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
40	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
41	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
42	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1.47
43	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
44	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1.29
45	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
46	S	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
47	S	1	1	1	2	3	1	1	1	1	2	2	1	1	1	1	1	1	1.05
48	P	1	1	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1
49	P	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

POST OPERATIVE STRAIN QUANTIFICATION

Patient number	BA	BAS	BIS	BI	BIL	BAL	MA	MAS	MIS	MI	MIL	MAL	AA	AS	AI	AL	A	A4C	A2C	A3C	GLS postb op
1	15-	11-	N	25-	23-	N	22-	27-	N	9-	5-	N	18-	N	16-	21-	18-	12-	17-	14-	-15
2	12-	11-	17-	17-	18-	10-	20-	25-	10-	12-	28-	23-	19-	23-	17-	23-	21-	17-	14-	21-	-17
3	N	9-	5-	N	19-	25-	N	15-	13-	17-	13-	17-	19-	19-	N	26-	23-	24-	12-	13-	-11
4	12-	12-	15-	10-	18-	4-	17-	20-	7-	12-	N	22-	3-	17-	18-	14-	17-	11-	8-	8-	-9
5	11-	13-	19-	14-	17-	8-	13-	17-	12-	10-	16-	8-	17-	13-	8-	3	5-	10-	11-	9-	-7
6	12-	14-	18-	13-	11-	24-	13-	9-	12-	12-	10-	25-	23-	24-	23-	10-	20-	20-	15-	11-	-14
7	12-	12-	12-	27-	21-	12-	28-	31-	18-	17-	27-	9-	24-	16-	26-	17-	22-	10-	27-	23-	-20
8	9-	N	8-	19-	N	4-	11-	N	8-	15-	N	11-	4-	6-	15-	15-	9-	8-	5-	7-	-17
9	19-	N	2-	4-	N	14-	10-	N	21-	12-	19-	3-	12-	22-	21-	19-	17-	14-	12-	10-	-13
10	5-	17-	8-	16-	11-	4-	7-	14-	13-	9-	6-	9-	10-	9-	8-	3-	8-	8-	7-	3-	-6
11	11-	7-	15-	23-	18-	6-	18-	28-	9-	15-	11-	23-	20-	16-	15-	21-	18-	14-	16-	16-	-15
12	13-	10-	16-	17-	28-	14-	12-	15-	18-	16-	13-	19-	23-	20-	23-	6-	20-	11-	17-	17-	-15
13	14-	8-	20-	30-	11-	24-	18-	13-	13-	15-	18-	32-	26-	18-	24-	12-	19-	17-	20-	12-	-17
14	6-	10-	6-	9-	28-	18-	6-	13-	17-	10-	10-	15-	12-	19-	8-	11-	11-	12-	7-	12-	-10
15	19-	13-	12-	22-	28-	26-	16-	27-	28-	20-	25-	17-	36-	19-	20-	27-	26-	23-	20-	32-	-26
16	24-	10-	14-	28-	13-	23-	22-	14-	23-	28-	15-	21-	31-	29-	29-	22-	27-	25-	2-	14-	-22
17	18-	13-	21-	18-	18-	24-	16-	25-	26-	22-	13-	22-	25-	35-	22-	22-	27-	25-	21-	19-	-22
18	23-	15-	17-	23-	28-	22-	11-	22-	14-	15-	17-	12-	28-	13-	24-	29-	28-	16-	27-	24-	-22
19	5-	13-	21-	18-	18-	8-	11-	8-	8-	15-	2-	11-	28-	14-	13-	17-	18-	9-	11-	21-	-14
20	24-	13-	11-	28-	21-	11-	13-	14-	15-	19-	11-	9-	24-	24-	33-	25-	25-	12-	24-	12-	-16
21	17-	13-	15-	15-	11-	18-	14-	18-	12-	15-	19-	16-	10-	21-	17-	24-	19-	21-	13-	13-	-15
22	8-	8-	20-	9-	13-	12-	13-	17-	11-	16-	9-	11-	27-	30-	30-	21-	26-	19-	19-	14-	-17
23	12-	5-	6-	7-	12-	8-	13-	1-	13-	18-	16-	17-	31-	18-	13-	23-	22-	12-	12-	16-	-13
24	9-	16-	11-	8-	12-	14-	10-	17-	11-	23-	11-	15-	14-	24-	11-	1-	10-	13-	7-	9-	-10
25	5-	8-	11-	10-	8-	7-	7-	4-	9-	11-	4-	6-	11-	11-	16-	14-	13-	9-	9-	8-	-9
26	10-	12-	18-	18-	22-	3-	9-	7-	5-	10-	8-	8-	13-	6-	16-	9-	10-	8-	11-	9-	-9
27	N	13-	13-	N	18-	18-	N	18-	15-	N	13-	13-	20-	9-	N	13-	14-	8-	12-	13-	-13
28	13-	13-	16-	8-	8-	24-	9-	11-	11-	12-	11-	23-	13-	16-	7-	2-	6-	8-	8-	8-	-8
29	16-	5-	10-	9-	13-	15-	16-	24-	24-	25-	21-	13-	19-	24-	29-	13-	19-	11-	19-	16-	-15
30	22-	10-	8-	7-	16-	13-	16-	30-	26-	16-	14-	15-	16-	16-	21-	26-	20-	18-	16-	19-	-18
31	12-	7-	13-	30-	13-	18-	25-	18-	14-	29-	18-	11-	22-	23-	35-	20-	23-	16-	25-	16-	-19
32	18-	13-	2-	8-	22-	20-	18-	30-	16-	13-	15-	19-	21-	16-	10-	25-	18-	13-	7-	19-	-13
33	9-	8-	21-	12-	7-	10-	10-	2-	14-	11-	22-	16-	25-	22-	30-	25-	25-	18-	19-	17-	-18
34	13-	7-	13-	16-	27-	10-	19-	25-	19-	27-	19-	15-	23-	11-	27-	24-	24-	11-	23-	18-	-20
35	9-	N	11-	12-	N	12-	11-	9-	14-	N	14-	17-	23-	17-	16-	18-	12-	12-	14-	12-	-12
36	19-	6-	N	21-	16-	N	20-	19-	N	16-	12-		16-	N	16-	9-	14-	10-	12-	12-	-12
37	11-	6-	14-	17-	18-	3-	10-	11-	11-	8-	16-	10-	18-	16-	16-	10-	15-	9-	14-	12-	-12
38	N	8-	7-	8-	14-	9-	3-	4-	12-	8-	4-	12-	8-	8-	4-	N	2-	8-	5-	5-	-3
39	13-	13-	3-	16-	10-	15-	10-	30-	17-	6-	20-	9-	3-	6-	9-	13-	6-	6-	10-	13-	-6
40	9-	20-	24-	7-	28-	22-	11-	15-	20-	14-	13-	7-	19-	30-	17-	29-	24-	22-	11-	19-	-17
41	13-	12-	8-	18-	21-	24-	22-	2-	30-	30-	13-	1-	29-	32-	32-	21-	27-	25-	24-	18-	-22
42	13-	14-	11-	4-	22-	16-	8-	7-	12-	17-	13-	11-	21-	6-	12-	21-	17-	10-	12-	14-	-12
43	19-	8-	N	12-	5-	N	13-	10-	N	15-	16-		22-	N	31-	17-	23-	14-	16-	15-	-15
44	21-	13-	10-	15-	18-	22-	16-	19-	21-	23-	23-	9-	25-	25-	27-	16-	22-	18-	21-	17-	-18
45	13-	20-	6-	11-	10-	21-	10-	13-	19-	15-	5-	10-	9-	23-	13-	17-	14-	16-	10-	10-	-12
46	12-	N	20-	14-	N	N	7-	N	4-	15-	N	20-	13-	9-	14-	7-	10-	9-	10-	11-	-9
47	14-	11-	11-	18-	8-	13-	10-	13-	7-	14-	25-	14-	16-	15-	16-	11-	13-	11-	13-	9-	-11
48	15-	4-	8-	4-	7-	9-	10-	12-	13-	17-	15-	11-	0	11-	13-	10-	9-	10-	6-	10-	-9
49	17-	11-	12-	16-	**6-**	13-	12-	8-	19-	18-	15-	16-	18-	7-	7-	13-	12-	13-	15-	9-	-12
50	17-	13-	12-	22-	22-	11-	12-	12-	4-	16-	12-	15-	30-	32-	18-	17-	24-	15-	17-	17-	-16

POST OPERATIVE 2D ECHO PARAMETERS

patient no.	ESV(ml)	EDV(ml)	SV(ml)	CO(ml)	EF(%)
1	22	68	36	2.5	52%
2	27	78	51	4.2	67%
3	23	84	61	5.6	72%
4	17	77	60	5.3	78%
5	65	145	37	3.3	75%
6	20	54	34	3.1	62%
7	27	94	67	5.8	68%
8	28	103	75	5.4	72%
9	19	69	50	3.2	65%
10	17	62	45	4.8	74%
11	29	79	50	3.6	56%
12	28	67	39	2.6	66%
13	19	**65**	46	2.6	78%
14	30	86	56	5	52%
15	27	96	69	5.2	65%
16	24	78	56	5.3	68%
17	23	62	39	2.4	62%
18	26	84	58	5.8	70%
19	17	51	34	3.1	66%
20	8	41	48	4	80%
21	23	56	50	4.5	61%
22	19	49	30	2.4	61%
23	21	89	68	5.8	76%
24	23	67	44	3.9	58%
25	30	64	34	2.9	53%
26	33	65	32	2.9	50%
27	22	72	50	5.7	70%
28	21	58	37	6	63%
29	16	54	38	4	70%
30	24	68	44	4.2	67%
31	25	71	46	4.2	62%
32	24	97	73	6.1	56%
33	31	88	57	5.2	55%
34	15	76	61	3.4	70%
35	19	77	58	5.5	68%
36	19	66	46	4	66%
37	28	67	39	3.2	54%
38	58	98	57	3.9	55%
39	15	68	53	3.2	67%
40	20	65	45	4	65%
41	21	73	52	4.4	75%
42	23	82	59	4.7	70%
43	11	35	24	4.5	69
44	15	44	29	2.6	65
45	28	74	46	3.8	62

46	22	90	68	4.8	75
47	39	85	46	3.6	54
48	27	61	34	3	53
49	28	67	39	4.2	65
50	20	79	59	4.1	60%

SURGICAL AND CPB PARAMETERS

Patient No	Number of grafts	CPB time(min)	Aortic cross clamp time (min)	Surgeons graft satisfaction score	perfusion pressure(mmHg)	CPB flow(L/min)
1	4	98	49	GOOD	55-60	3.8-5
2	4	90	46	GOOD	50-70	3.5-4.5
3	4	105	43	GOOD	50-70	3.5-4.5
4	3	110	55	GOOD	60-70	3.5-4
5	5	105	65	GOOD	45-65	3.5-4
6	4	129	60	GOOD	45-65	3.8-5
7	5	97	60	GOOD	60-70	2.8-3.5
8	4	95	49	GOOD	50-70	3.0-5.0
9	3	100	44	GOOD	55-60	3.5-4.5
10	5	89	49	GOOD	45-65	3.5-4.5
11	5	94	55	GOOD	60-70	3.8-5
12	5	120	53	GOOD	55-60	3.5-4.5
13	4	130	44	GOOD	55-60	3.5-4.5
14	5	129	48	GOOD	60-70	3.5-5.0
15	4	98	43	GOOD	45-65	3.8-5
16	4	80	34	Adequate	50-70	2.5-3
17	3	112	33	GOOD	50-70	3.5-4.5
18	5	117	55	GOOD	60-70	2.8-4.0
19	5	100	59	GOOD	55-60	3.8-5
20	4	105	49	GOOD	50-70	3.5-4.5
21	5	105	51	GOOD	60-70	2.8-3.5
22	5	134	53	GOOD	55-60	3.5-4.5
23	4	132	44	GOOD	60-70	2.8-4.0
24	4	105	50	GOOD	60-70	3.5-4.5
25	5	112	49	GOOD	50-70	3.0-4.0
26	4	98	55	GOOD	45-65	3.8-5
27	4	89	51	GOOD	60-70	2.8-3.5
28	4	84	53	GOOD	55-60	3.5-4.5
29	5	109	67	GOOD	45-65	3.5-4.5
30	4	97	49	GOOD	55-70	3.0-4.0
31	4	114	44	GOOD	60-70	3.8-5
32	4	105	54	GOOD	50-70	2.8-4.0
33	5	102	58	GOOD	60-70	2.8-3.5
34	3	107	38	GOOD	45-65	3.0-4.0
35	4	112	49	GOOD	50-70	2.8-4.0
36	3	106	39	GOOD	45-65	3.8-5
37	4	96	49	GOOD	60-70	3.0-4.0
38	4	99	45	GOOD	50-70	3.5-4.5
39	4	102	50	GOOD	50-70	2.8-3.5
40	4	112	49	GOOD	45-65	3.8-5
41	4	120	48	GOOD	50-70	2.8-4.0
42	4	130	50	GOOD	45-65	3.0-4.0
43	4	114	53	GOOD	50-70	3.5-4.5
44	5	108	62	GOOD	45-65	3.8-5
45	5	98	50	GOOD	50-70	3.5-4.5
46	3	93	52	GOOD	50-70	2.8-3.5
47	4	99	50	GOOD	45-65	2.8-4.0
48	3	108	45	GOOD	50-70	3.0-4.0
49	4	117	55	GOOD	50-70	2.4-4.0
50	4	120	60	GOOD	50-60	2.5-5-4

POST OPERATIVE PARAMETERS

Patient no.	inotrope 1	inotrope 2	inotroopic score	duration OF INOTROPIC USE (min)	mechanical ventilation duration (HRS)	ICU stay duration (HRS)	Ph	Hemoglobin	BE/BD
1	ADR		5	140	18	72	7.4	9.8	-2.4
2	ADR		5	180	12	48	7.33	9	-3
3	ADR	NOR-ADR	10	350	10	72	7.32	13.2	2
4	ADR		5	170	12	48	7.48	12.8	-2
5	ADR	NOR-ADR	20	270	20	72	7.35	12	-1.5
6	ADR		5	120	16	48	7.32	12.3	-0.3
7			0		14	48	7.35	13.4	-0.4
8	ADR	NOR-ADR	10	450	12	48	7.31	11	-1
9	ADR	NOR-ADR	10	400	10	48	7.39	11.1	3
10	ADR		5	140	8	48	7.39	13	-3
11	ADR	NOR-ADR	7.5	380	16	48	7.37	14.5	-2.8
12	ADR	NOR-ADR	10	320	20	44	7.33	14	1.2
13	ADR	NOR-ADR	10	390	8	44	7.4	12.3	-4
14	ADR	NOR-ADR	10	470	12	45	7.28	11	-2.3
15	ADR	NOR-ADR	10	500	18	42	7.31	11.4	1.7
16	ADR		5	240	15	24	7.37	11.3	-2
17	ADR		5	200	7	40	7.4	11	0
18	ADR	NOR-ADR	12.5	320	11	38	7.43	9.7	-0.9
19	ADR		7.5	300	10	40	7.32	9	-1
20	ADR	NOR-ADR	12.5	470	15	42	7.3	12	-0.4
21	ADR	NOR-ADR	12.5	320	13	35	7.39	10.7	-1.3
22	ADR	NOR-ADR	10	280	12	38	7.44	10	-1.4
23	ADR		5	200	4	39	7.39	9.4	-1.7
24	ADR		2.5	210	5	33	7.49	9.7	-1.8
25	ADR	NOR-ADR	5	250	6	35	7.4	8.9	1.4
26	ADR	NOR-ADR	15	390	11	37	7.42	10.7	-1
27	ADR		5	300	10	38	7.33	12.9	0.7
28	ADR		5	340	11	36	7.32	11.6	-0.5
29	ADR	NOR-ADR	10	420	12	40	7.34	10.7	1.2
30	ADR		2.5	240	14.5	38	7.37	12.3	-0.7
31	ADR		2.5	240	11	41	7.34	11.7	1.3
32	ADR	NOR-ADR	10	340	10	42	7.32	10.7	1.9
33	ADR		5	100	11	40	7.34	11.2	-3
34					7	42	7.38	10.7	-3.4
35	ADR	NOR-ADR	5	440	11	39	7.33	11.5	2.1
36	ADR	NOR-ADR	10	440	15	33	7.32	10.5	-2.4
37					6	39	7.31	9.7	2.6
38	ADR	NOR-ADR	5	400	10	35	7.34	8.8	1.3
39	ADR		5	230	9	38	7.35	9.9	-1
40	ADR	NOR-ADR	10	300	10	40	7.35	14	0.9
41	ADR	NOR-ADR	10	400	12	40	7.34	11.8	0.7
42	ADR		5	200	9	40	7.35	11.6	-0.6
43	ADR	NOR-ADR	10	380	13	38	7.33	10	1.8
44	ADR		5	230	9	37	7.47	11	-2.6
45	ADR	NOR-ADR	5	280	7	39	7.42	9.8	-3.2
46	ADR		5	180	6	35	7.32	11	-4
47	ADR	NOR-ADR	7.5	380	11	40	7.35	10.6	-2.5
48	ADR		5	250	9	45	7.33	8.6	3.2
49	ADR	NOR-ADR	10	300	24	72	7.35	10.2	-2.6
50	ADR		7.5	200	16	48	7.32	9.4	-3.2