EFFECT OF SEVOFLURANE VERSUS PROPOFOL ON STRAIN QUANTIFICATION IN PATIENTS WITH SEVERE AORTIC STENOSIS UNDERGOING AORTIC VALVE REPLACEMENT

Dr. MARKOSE L PARET

DM CARDIOTHORACIC AND
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A THESIS SUBMITTED BY

DR. MARKOSE L PARET

TO

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM.

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE AWARD OF

DM CARDIOTHORACIC AND VASCULAR ANESTHESIOLOGY

YEAR 2020 TO 2023

DECLARATION BY THE STUDENT

CERTIFICATE

I, Dr. Markosé L Paret hereby certify that I had personally carried out the work depicted in the thesis titled,

"Effect of sevoflurane versus propofol on strain quantification in patients with severe aortic stenosis undergoing aortic valve replacement",

No part of this thesis has been submitted for the award of any other degree or diploma prior to this date.

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Date 20.8.2022

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20 August 2022 Dr Markose L Paret

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LIST OF ABBREVIATIONS

SL NO	ABBREVIATION	FULL FORM
1.	AS	Aortic stenosis
2.	LVEF	Left ventricular ejection fraction
3.	LV	Left ventricular
4.	AVR	Aortic valve replacement
5.	ARB	Angiotensin receptor blocker
6.	ACEI	Angiotensin converting enzyme inhibitor
7.	CCB	Calcium channel blocker
8.	ME	Mid oesophageal
9.	TG	Trans gastric
10.	2Ch	Two chamber
11.	3Ch	Three chamber
12.	4 Ch	Four chamber
13.	LAX	Long axis
14.	SAX	Short axis
15.	2D STE	Two-dimensional speckle tracking echocardiography

LIST OF ABBREVIATIONS

SL NO	ABBREVIATION	FULL FORM
16.	TTE	Transthoracic echocardiography
17.	TEE	Transoesophageal echocardiography
18.	GLS	Global longitudinal strain
19.	GCS	Global circumferential strain
20.	GRS	Global radial strain
21.	ASE	American society of echocardiography
22.	JASE	Journal of American society of echocardiography
23.	EACVI	European association for cardiovascular imaging
24.	TDI	Tissue doppler imaging
25.	CMRI	Cardiac magnetic resonance imaging
26.	ROI	Region of interest
27.	RCT	Randomized controlled trial
28.	BIS	Bispectral index
29.	CPB	Cardiopulmonary bypass
30.	VIS	Vasopressor inotrope score

SYNOPSIS

EFFECT OF SEVOFLURANE VERSUS PROPOFOL ON STRAIN QUANTIFICATION IN PATIENTS WITH SEVERE AORTIC STENOSIS UNDERGOING AORTIC VALVE REPLACEMENT

SYNOPSIS

BY

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SYNOPSIS

Background:

Severe AS with good LVEF is known to have abnormal 2D STE strain values in preoperative period that changes after aortic valve replacement. There are scarce studies elaborating changes in strain values in immediate post valve replacement period. This study evaluated changes in GLS and GCS values post-AVR in perioperative period. In our study, we analyzed changes in GLS and GCS values after AVR in patients of severe AS with LVEF more than 50%. Comparison was also done between effects of sevoflurane anesthesia regimen and propofol anesthesia regimen on changes in GLS and GCS values post-AVR. Our study also evaluated pre-CPB GLS and GCS values in these patients together with correlation of preoperative risk factors like hypertension, diabetes, ARB/ACEI usage, CCB usage and beta blocker usage with pre-CPB GLS and GCS values.

Materials and Methods:

Our study recruited severe AS patients in sinus rhythm with good LVEF (LVEF > 50%) more than 18 years of age undergoing elective AVR. Patients with other conditions that may affect strain values were excluded from study together with patients with poor echocardiographic wall differentiation and contraindications to TEE. Study was designed as prospective, randomized study for which institutional ethics committee approval was obtained. After obtaining informed written consent, 22 patients in Group-S received sevoflurane anesthesia regimen and 21 patients in Group-P received propofol anesthesia regimen as per study protocol. 2D STE GLS, GCS and regional strain values were acquired in both groups as per study protocol. Study data like strain values, patient demographics, preoperative conventional echocardiographic parameters, surgical parameters, CPB parameters, VIS and

postoperative conventional echocardiographic parameters were analyzed. Intra-observer and inter-observer variability was assessed with intra-class and inter-class correlation coefficient.

Results and observations:

Propofol group was found to be having higher GLS than sevoflurane group in pre-CPB period (mean ± SD of propofol pre-CPB GLS -12.56 ± 2.36 vs sevoflurane pre-CPB GLS -10.41 ± 2.46). There were no other differences between both groups in pre-CPB and post-CPB period. It was observed that GCS decreased across both groups post-AVR (mean ± SD of pre-CPB GCS -25.28 ± 3.67 vs post-CPB GCS -22.98 ± 4.01). However, there were no changes observed in GLS across all patients after AVR. Intragroup analysis of pre-CPB and post-CPB strain values in sevoflurane group revealed a significant reduction in GCS (mean ± SD of sevoflurane pre-CPB GCS -26.18 ± 3.97 vs sevoflurane post-CPB GCS -22.95 ± 3.97). There were no similar differences in strain values in propofol group on intragroup analysis. No differences were found between sevoflurane and propofol groups in mean of change in GLS and GCS values after AVR. A low GLS and high GCS were observed in all patients of severe AS with good LVEF in pre-CPB period, when compared to published normal ranges. Pre-operative CCB usage was discovered to be associated with higher pre-CPB GCS. Similarly, patients on ARB/ACEI had low GLS in pre-CPB period. There was weak negative correlation ($r = - 0.29$) between VIS on arrival in ICU and pre-CPB GLS. A weak positive correlation ($r = 0.277$) was observed between VIS on arrival in ICU and pre-CPB GCS. Intra- class and inter-class correlation was found to be good on analysis (Intra- class coefficient and inter-class coefficient 0.7- 0.9).

Conclusion:

In the pre-CPB period, the GLS was found better in propofol group than sevoflurane group. Post-CPB GCS was less than pre-CPB GCS across study groups. When pre-CPB GCS were compared with post-CPB GCS in sevoflurane group, we observed that the GCS value reduced in sevoflurane group. When we evaluated for changes in the GLS and GCS in sevoflurane group with those of propofol group, we found no statistically significant difference. GLS values across study groups in the pre-CPB period were less, whereas the GCS values were increased, when compared to published normal range. Patients who were on CCB had increased GCS values pre-CPB when compared to those not on them. Patients on ACEI/ARB were found to be having significantly less GLS score preoperatively when compared to those not using them.

INTRODUCTION

INTRODUCTION

Perioperative transesophageal echocardiography (TEE) is routinely done by cardiac anesthesiologists for real time evaluation of cardiac function during cardiac surgery.

Two dimensional speckle-tracking using echocardiographic imaging allows the angle-independent evaluation of myocardial strain in the three directions (longitudinal, circumferential and radial), providing comprehensive information on left ventricular (LV) myocardial contractility (1) and is considered better than other 2D based calculations of LV systolic function like ejection fraction (EF).

Patients having severe aortic stenosis (AS) with preserved left ventricular ejection fraction (LVEF) may have subclinical myocardial dysfunction possibly due to subendocardial ischemia, which is characterised by impaired global longitudinal strain (GLS) (2). In asymptomatic patients of severe AS with preserved ejection fraction, strain imaging has also frequently revealed low longitudinal strain with supranormal circumferential strain (3).

Studies evaluating the effect of aortic valve replacement (AVR) on strain values during postoperative follow up period have shown an increase in longitudinal strain and decrease in circumferential strain compared to preoperative values (1,3). As there is a drastic reduction in LV afterload after AVR in these patients, changes seen in the echocardiographic strain values in postoperative follow-up period may also be expected to be encountered in the early postoperative period.

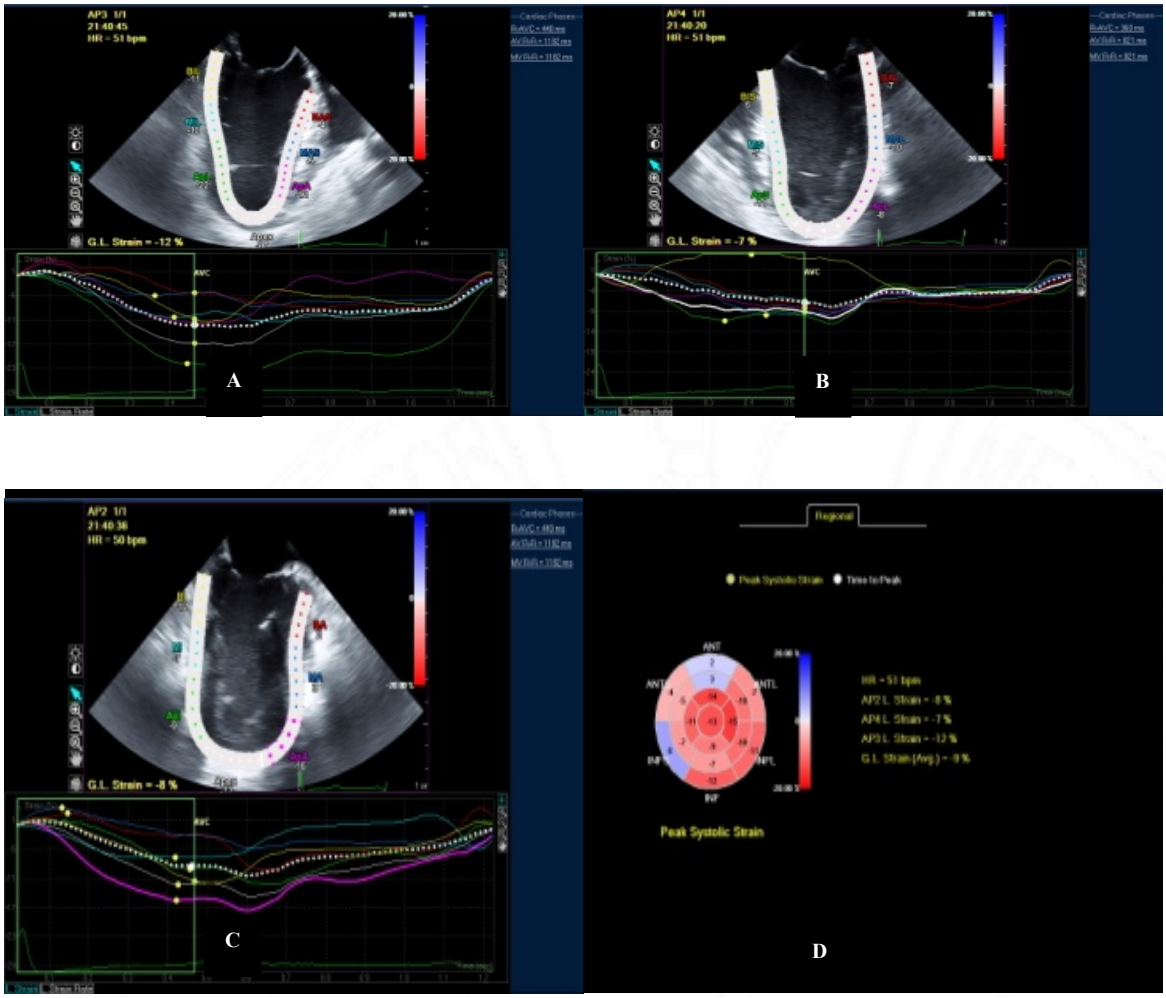


Figure 1: Calculation of longitudinal strain from Mid oesophageal three chamber (A), four chamber (B) and two chamber (C) views with bulls eye image (D) showing global longitudinal strain.

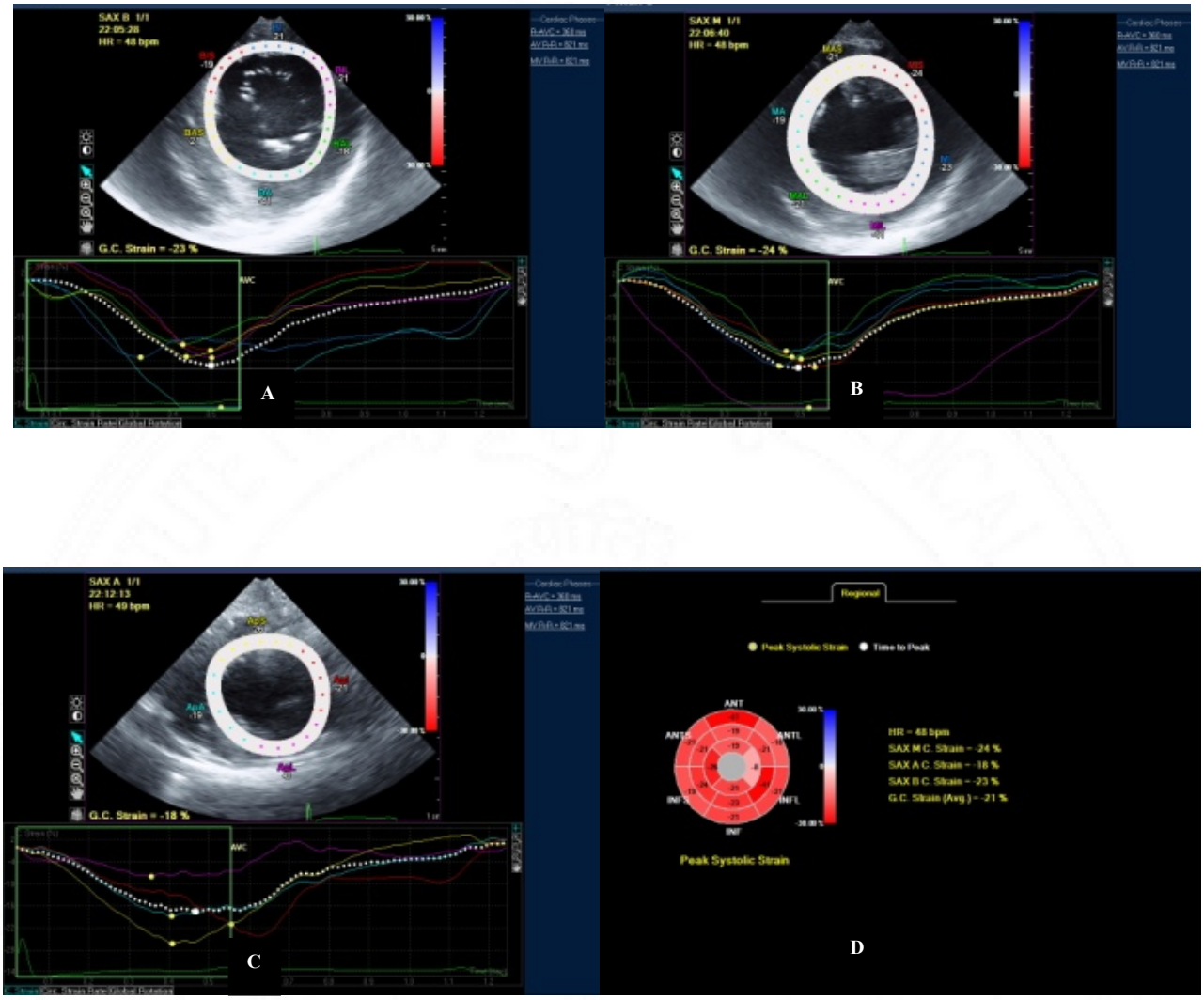


Figure 2: Calculation of circumferential strain from basal (A), mid (B) and apical (C) transgastric short axis views and bulls eye image (D) depicting global circumferential strain.

Anesthesia for AVR is administered commonly using sevoflurane-based technique or propofol infusion technique. It is debatable as to which anesthetic agent of these two, provides better myocardial protection in the setting of cardiac surgery**.** Both the anesthetic regimens employed in cardiac surgery produce different effect on strain quantification(4,5). Since both the regimens are known to be myocardial suppressants, we included patients having good LV systolic function in the study groups. We conducted a study to compare effects of sevoflurane and propofol anesthesia regimens on 2D speckle tracking imaging before and after AVR in patients of severe AS having good LVEF.

Our study estimated 2D speckle tracking echocardiography (STE) strain values in patients of severe aortic stenosis with good LV function prior to and succeeding to AVR before and after cardiopulmonary bypass (CPB). We also analyzed the preoperative risk factors associated with deranged strain values before surgery.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES OF THE STUDY

Primary objectives:

1. To study intraoperative changes in global longitudinal strain (GLS) and global circumferential strain (GCS) after AVR in patients of severe AS with good LVEF.

2. To compare effects of sevoflurane and propofol anesthesia regimens on changes in GLS and GCS after AVR in same study subjects.

Secondary objectives:

1. To assess the preoperative baseline values of GLS and GCS in patients with severe AS and good LVEF.

2. To study preoperative risk factors which have significant influence in altering the preoperative GLS and GCS values in same study subjects.

LITERATURE REVIEW

REVIEW OF LITERATURE

The present echocardiographic techniques for assessment of LV systolic function during intraoperative period and its constraints

Accurate assessment of LV systolic function is an important aspect of perioperative echocardiography in AS patients as it helps in deciding the optimal timing for valve replacement, especially in asymptomatic patients (6,7). In addition, it also identifies the subset of patients having a poor prognosis, even after AVR (8,9).

There are several methods that are utilized for evaluation of global and regional left ventricular systolic function that ranges from 2DE- based methods such as visual assessment of thickening of walls and inward endocardial motion, fractional area change, fractional shortening, stroke volume, LVEF to LV 3D ejection fraction.

The commonest used technique for evaluation of global LV systolic function is LV ejection fraction. LVEF is an important determinant of classification, prognosis and mortality in patients with systolic heart failure and a key decision making factor in day-to-day clinical practice (10). It is commonly used due to its simplicity and is recommended by the American society of echocardiography (ASE) for assessment of intraoperative LV systolic function (11). However, it has many limitations such as load dependency and necessity for accurate tracing of endocardial border (12). LV ejection fraction calculation is also based on certain LV geometrical assumptions which may not be uniformly applicable in all patients (12). The biggest disadvantage with the use of LVEF in aortic stenosis patients is that it is a very later marker of LV systolic dysfunction in the progression of the disease(7).

The commonest technique used for evaluation of regional LV systolic function is scoring based on regional wall motion abnormalities (RWMA) according to ASE guidelines (11). This technique is also highly operator dependent.

Other commonly used techniques such as cardiac output measurement, stroke volume, fractional area change and fractional shortening are all load dependent and vary widely with operator experience and interpretation.

New and advanced 3D echocardiography protocols for evaluation of LV systolic function are expected to effectively override most of the technical issues faced by 2D echocardiography. However, they are limited by the need for high quality images, less temporal and spatial resolution of acquired images and advanced training required in echocardiography (13).

Need for correct estimation of LV systolic function in aortic stenosis patients

Aortic valve replacement is one of the commonest valve replacement procedures, amounting to 1,30,617 cases done in year 2019 in US alone (14). Transoesophageal echocardiography is an essential tool in evaluation of LV systolic function in these patients. Current methods to measure LV systolic function depends heavily on echocardiographic calculations like LV ejection fraction and cardiac output. These techniques fail in early detection of LV systolic dysfunction in aortic stenosis patients thereby affecting timely intervention and decision making in these patients (12).

Accurate detection of LV systolic dysfunction helps in timely replacement of the valve in AS, which in turn results in better prognosis and less mortality (6).

Correct evaluation of LV systolic function using LVEF is also important in stratification of symptomatic low-flow low-gradient severe AS into those with low ejection fraction (D2) and with normal ejection fraction (D3) which differ in their therapeutic management (6,15).

Myocardial deformation and strain imaging and its importance in aortic stenosis

Myocardial fibers are organized in a layered fashion with a leftward helix pattern in subepicardium and rightward helix pattern in subendocardium. Such a pattern leads to different cardiac motions in longitudinal, circumferential, and radial plane during systole and diastole. Longitudinal shortening, circumferential thinning and radial thickening occurs in systole which results in clockwise LV rotation at base and anti-clockwise LV rotation at apex when seen from apical end (Figure 3).

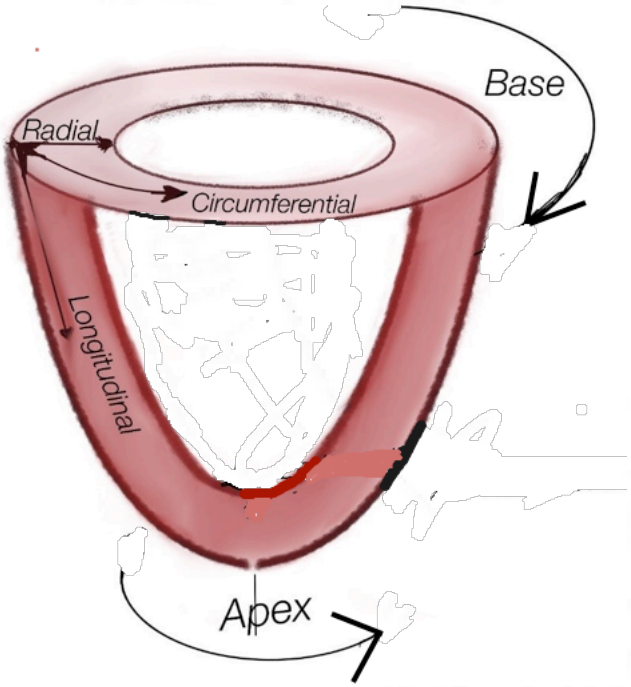


Figure 3: Various strain motions in systole

Such an intricate 3D cardiac motion of various layers of fibers is not appreciated with commonly used 2D TEE imaging. 2D echocardiography images point towards functionality of middle layer of LV myocardium, as inward excursion of endocardium in midesophageal views and radial thickening in transgastric views. Mathematical modelling studies have shown that circumferential shortening contributes to 67% and longitudinal shortening contributes to 33% of LV stroke volume during systole (16). Strain imaging using LV GLS has been shown to detect early changes in left ventricular systolic and diastolic function when compared with commonly used variables such as LVEF (17).

GLS decreases together with a compensatory increase in GCS in patients with severe AS with good LVEF (3). These changes are not reflected with corresponding changes in LVEF in early stages of the LV systolic dysfunction.

Researchers have also found changes in GLS and GCS after aortic valve replacement in patients with severe aortic stenosis and good LV systolic function during follow up in outpatient clinics (1,3,18,19). However, there is a no literature regarding changes in LV strain parameters immediately after AVR.

2D speckle tracking echocardiography (2D STE)

There are many existing techniques that analyze LV strain such as 2D STE, cardiac MRI (CMR) and tissue doppler imaging (TDI) (20). 2D STE and TDI are the commonly available techniques for strain echocardiography. 2D STE aids in evaluation of global and segmental longitudinal, circumferential, radial strain and strain rate together with LV rotation, torsion and dyssynchrony (21).

STE utilizes randomly generated artefacts due to reflection, refraction and scattering in echo images. Such artefacts, known as speckles, are used as natural acoustic markers for evaluation of myocardial motion using specialized software. This software recognizes a 'cluster of speckles' known as 'kernel' and monitor them frame-by-frame during cardiac cycle. Strain imaging software calculates strain and strain rate from speckle tracking of the concerned myocardial segment and gives a consolidated global value for longitudinal and circumferential strain (21).

The advantage of STE-derived strain value vis a vis TDI is that it is angle independent, as STE utilizes measurement of displacement of region of interest (ROI) when compared with baseline images over a specific cardiac cycle. STE calculates deformation simultaneously in two directions like transverse and longitudinal direction from LAX views and circumferential and radial direction from SAX views of TEE. Translation and tethering have least effect on speckle tracking strain (21). LV strain quantification using STE was found to be associated with least intra-observer and inter-observer variability and hence, can be regarded as a reliable method for assessment of LV myocardial strain (21)

Drawbacks in strain quantification using STE

The main limitation with 2D STE is its dependance on good image quality (21). The ability of software to track endocardial borders and speckle pattern is mainly based on good quality 2D echocardiography images.
In a study conducted in healthy normal subjects , about 6% of all myocardial segments could not be assessed due to poor image quality (22).

Speckle tracking strain quantification is also influenced by complex out of plane motion by heart in 3D plane, making frame-by-frame speckle tracking difficult (21). Reverberation artifacts and acoustic shadowing also

interfere with STE, affecting precision strain measurement.

Semi-automated software is used in STE wherein the operator places speckles at ROI and defines myocardial tracking prior to processessing of strain values. This could also result in possible wide range of strain values and considerable intraobserver variability.

STE lacks appropriate grading due to scarcity of published literature and because of use of various quantification softwares by different vendors. EACVI and ASE have liaised with industry partners and brought out a consensus document defining common standards for 2D STE measurements (23). This is expected to create a uniform pattern across all vendor platforms in calculating strain values, thereby providing reference values for different types of strain used in clinical scenario.

Despite all these limitations, STE strain quantification remains a convenient technique that is angle-independent, consistent and can be carried out in intraoperative period.

Comparison of 2D LVEF with strain values derived from 2D STE

2D LVEF by Simpsons biplane method is the recommended echocardiographic technique for evaluation of LV systolic function by ASE (11). It has been compared with strain values derived from 2D STE in many studies. GLS derived from 2D STE is more sensitive than LVEF in detecting early LV systolic dysfunction (24). The GLS, when matched with 2D LVEF, has better reproducibility in evaluation of the LV systolic function (25). It holds true even in presence of hypertrophic cardiomyopathy with preserved LVEF (26). 2D STE has also been shown as being accurate in the identification of patients with 2D LVEF < 55% (27). To summarize, strain values derived from 2D STE when compared with 2D LVEF are accurate, reproducible and are better at evaluating LV systolic function in a remodeled myocardium. Thus, it justifies performing 2D STE derived strain analysis in conjunction

with 2D LVEF for routine evaluation of LV systolic function and also employing it as an effective tool in ascertaining early LV systolic dysfunction (28).

Comparison of 2D STE derived strain with reference method for evaluation of LV systolic function

CMR is the existing gold standard for evaluation of volumetric LV systolic function (29). Brown et al (30) in his study found that GLS was in agreement with cardiac MRI EF for evaluation of LV systolic function. Onishi et al (31) evaluated 2D STE derived strain values with strain values derived from feature tracking CMRI and CMRI EF and concluded that 2D STE derived strain values were closely related with feature tracking CMRI strain values and both correlated well with cardiac MRI derived EF.

Published literature on intraoperative 2D STE for estimation of LV systolic function

There is scant literature on intraoperative use of 2D STE for evaluation of LV systolic function. Chennakeshavullu et al (4) compared sevoflurane and propofol-based anesthesia regimens in coronary artery bypass grafting (CABG) patients and found that there was no significant difference in GLS between the groups. Zhang et al (32) ascertained efficacy of intraoperative TEE-derived GLS, GCS and global radial strain (GRS) to predict outcomes after AVR and reported that the GLS can predict prolonged hospitalization and need for increased postoperative need for inotropic support . Labus et al (33) observed serial changes in the intraoperative values of GLS in CABG patients and found that GLS was not significantly different between awake patients and patients under anesthesia. They also noticed that GLS values did not reduce after sternotomy although, they worsened after CABG despite postoperative period remaining uneventful.

Effect of Volatile anesthetic agents or Propofol on myocardial protection

Sevoflurane-based or propofol-based regimens are the two widely used anesthetic techniques in cardiac surgery. Studies comparing the myocardial protective effects of these regimens have yielded equivocal results (34–42). It is hypothesized that sevoflurane provides better myocardial protection during cardiac surgery due to its anesthetic preconditioning effects (43). De Hert et al (34) in his study comparing sevoflurane with propofol in cardiac surgery reported that cardioprotective effect of sevoflurane were associated with less increase in biomarkers of myocardial damage in first 36 postoperative hours. The same group (35), on further comparison of the sevoflurane and the desflurane with propofol in elderly high-risk patients undergoing cardiac surgery noticed that these inhalational agents were better in preserving the myocardial function in the postoperative period. On the contrary, in another study (36) the authors administered either isoflurane or propofol in conjunction with opioids in patients undergoing CABG and discovered that volatile-based technique did not offer any additional benefit over propofol-based technique in terms of reducing the postoperative troponin levels. A similar study conducted by Poonam et al (37) comparing the effect of desflurane with propofol revealed no difference in overall outcome between groups. Landoni et al (38) conducted a multicenter randomized trial to compare sevoflurane and propofol in high risk cardiac surgery patients and observed that there was no advantage of sevoflurane on prolonged ICU stay or mortality. A study done on perioperative ischemia in major non-cardiac surgery patients by Giovanna et al (39) showed that sevoflurane when compared with propofol did not reduce the incidence of myocardial ischemia.

Several metanalysis have also been undertaken to compare these both anesthesia techniques in cardiac surgery. Feng Li et al (40) did analysis of 15 randomized controlled trials (RCT), evaluating cardioprotective effects of sevoflurane with propofol in cardiac surgery and found that sevoflurane is more cardioprotective than propofol. Another meta-analysis (41) of 37 RCT's noted that in adults undergoing cardiac surgery with cardiopulmonary bypass, volatile anesthetic agents were superior to propofol with respect to long term mortality

(5)and myocardial protection. A similar meta-analysis done recently (42) on 40 RCT's comparing sevoflurane with propofol in cardiac surgery found no difference between both groups on mortality, postoperative cardiac biomarker levels at 24 hour and time to tracheal extubation.

Effect of Sevoflurane and Propofol on 2D STE derived strain values

There are limited studies evaluating effect of sevoflurane or propofol on 2D STE derived strain values. Chennakeshavullu et al (4) utilized intraoperatively derived 2D STE strain values to compare sevoflurane and propofol based anesthesia regimens in CABG patients and found that there is no significant difference in GLS between both groups. Another study (5) analyzed the effect of propofol based anesthesia induction on GLS in laparoscopic cholecystectomy patients and discovered that GLS values did not reduce with propofol induction of anesthesia.

2D STE derived strain values in early postoperative period

Carasso et al (3) followed up myocardial mechanics using STE in severe AS with good LVEF and reported that GLS increased with a concurrent decrease in GCS in the early postoperative period after AVR (7+/- 3 days postoperatively). In a study done in complication free on-pump CABG patients (33) , it was observed that GLS worsened after surgery (11.75 +/- 5 days postoperatively). Further, postoperative follow-up at 17 months post AVR in severe AS with preserved LVEF (1) found that GLS , GCS and GRS improved during late follow-up period . However, to the best of our knowledge, we did not come across any other study on evaluation of STE values in immediate post AVR period.

Justification of our study

Severe AS patients with good LVEF have deranged echocardiographic 2D STE strain values due to subendocardial ischemia induced by increased left ventricular afterload and wall stress. Impaired GLS is often found in severe AS patients with preserved LVEF. There is a compensatory increase in GCS in this subset of patients which aids in preserving ejection fraction. As the disease progresses, this compensatory mechanism is blunted, as GCS return to normal values, resulting in reduced LVEF. Hence both these strain values are of use in appropriate timing of AVR and in prognostication of post-surgical course.

Follow up studies using 2D STE in these patients following AVR, have shown improvement in strain values. However, as all these studies were conducted in the postoperative period with a spread over time of 7 days to 17 months, they did not provide any information on changes in strain imaging in the immediate postoperative period. As left ventricular afterload is reduced immediately after AVR, we expect much earlier changes to occur in the strain quantification in these patients. Despite this understanding, there are no international or national studies evaluating perioperative strain values in AVR to the best of our knowledge.

During cardiac surgery, inhalational anesthetic preconditioning has been found to protect the myocardium from injury. Similar cardio-protective benefits were also found to be offered by intravenous anesthetic drugs such as propofol through its antioxidant properties. It is still controversial as to which of these two agents is more beneficial in protecting the myocardium during cardiac surgical procedures. There are no studies nationally or internationally comparing the effects of sevoflurane-based anesthesia with propofol-based anesthesia on perioperative strain values during AVR.

In addition, no research on this topic in AVR has been previously conducted in our institute. Therefore, we performed this study in adult patients with severe AS and good LVEF undergoing AVR in our institute.

MATERIALS AND METHODS

MATERIALS AND METHODS

This prospective randomized study was conducted in a tertiary referral center, operating 1200 adult cardiac surgical patients annually. The study was approved by the institutional ethics committee prior to enrollment of study population.

Patient selection

All adults above 18 years of age admitted to the institute with severe AS for elective AVR were enrolled into the study once deemed eligible and after obtaining informed consent.

Inclusion criteria

All adults above 18 years of age, in sinus rhythm, admitted with severe AS with good LV function (LVEF more than or equal to 50%) for elective AVR were included in the study.

Exclusion criteria

1. Emergency surgeries.
2. Redo surgeries.
3. Patients not willing to participate in study.
4. More than mild AR, MR or TR.
5. Patients with coronary artery disease defined by lumen stenosis more than or equal to 50% in at least one of major epicardial coronaries on coronary angiogram.
6. Patients receiving preoperative inotropes.
7. Patients with poor echo differentiation of endocardial borders.

8. Contraindication to TEE probe placement like esophageal strictures, esophageal varices, esophageal tumors, 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.

Thesis acceptance committee and Institutional ethics committee approval

Approval from both committees were taken prior to starting of study. IEC approval was given as per IEC number SCT/IEC/1843/FEBRUARY/2022

Sample size selection

The sample size was calculated assuming the expected mean and standard deviation of the percentage change in longitudinal strain in the Sevoflurane group as ,**σ1** (39,15) and in the propofol group as , **σ0** (29,15), as per the previous study by Carasso S et al (3). The other parameters considered for sample size calculation included were 80% power of study and 5% two-sided alpha error. The required sample size was calculated using the following formula as proposed by Kirkwood BR et al (44).

Formula used for sample size calculation:

$$N = \frac{(u + v)^2(\sigma_1^2 + \sigma_0^2)}{(\mu_1 - \mu_0)^2}$$

N	= Sample size
μ_1 , μ_0	= Difference between the means (μ_1=39 and μ_0=29)
σ_1, σ_0	= Standard deviations (σ_1=15 and σ_0=15)
u	= Two sided percentage point of the normal distribution corresponding to 100 % - the power = 80%, u =0.84
v	= Percentage point of the normal distribution corresponding to the (two sided) significance level for significance level = 5%, v = 1.960

The required sample size as per the above-mentioned calculation was 35 in each group. To account for an exclusion after recruitment rate of a about 5%, another 2 subjects were added to the sample size in each group. Thus, the final required sample size was 37 subjects in each group.

We could not achieve the desired sample size per group due to Covid related OT closures and less number of cases in OT. Hence, we have evaluated 22 patients recruited so far in sevoflurane group and 21 patients recruited in propofol group for the analysis.

Randomization

Numbered, sealed, opaque envelopes were used to randomly allocate patients to one of our study groups: Sevoflurane group (Group - S) or propofol group (Group – P).

Informed consent

Informed consent in English or Malayalam was obtained from the patient before start of the surgery.

Anesthesia protocol

Patients included in the study were educated on the study in the presence of a witness. The witness was allowed to counterquestion the patient whether he/she has really understood the proposed study, of which he/she would be a part. An informed consent form was signed by patient or relative of the patient according to the institute protocol.

Patients undergoing adult elective cardiac surgery were consecutively recruited and randomized into two groups Group-S: Sevoflurane group or Group-P: Propofol group.

Patients were instructed to continue all medications excluding oral hypoglycemic drugs, insulin, angiotensin - converting enzyme inhibitors and angiotensin - receptor blockers. Angiotensin-converting enzyme inhibitors and Angiotensin - receptor blockers were stopped 48 hours prior to surgery. Insulin and oral hypoglycemic drugs were omitted on the morning of surgery. Oral diazepam 0.2 mg/kg was administered for sedation, the night before surgery.

The induction of general anesthesia in the Sevoflurane group (Group-S) consisted of fentanyl (5-10 mcg/kg), midazolam (0.1 mg/kg), pancuronium (0.1 mg/kg) and sleep dose of propofol (1-2 mg/kg). Sevoflurane anesthesia was targeted to minimum end-tidal concentration of at least 1 minimal alveolar concentration during complete procedure. Sevoflurane anesthesia was utilized while patient was on CPB also, with BIS values kept between ranges of 40 - 50. A calibrated vaporizer was utilized to administer sevoflurane while patient was on CPB. Fentanyl was utilized to keep mean arterial pressure (MAP) within 20% from baseline, ensuring MAP more than 65 mm Hg.

Total intravenous anesthesia was utilized in patients in propofol group (Group - P) during surgery. Anesthesia induction was done with sleep dose of propofol (1-2 mg/kg), fentanyl (5-10 mcg/kg), midazolam (0.1 mg/kg) and pancuronium (0.1 mg /kg). Continuous infusion of propofol (50 -100μg/kg/min) was administered throughout the procedure including CPB. Fentanyl boluses (totaling to a dose of 20 mcg/kg/min) were utilized intermittently to ensure MAP within 20% from baseline and more than 65mmHg. Bispectral index (BIS) value of 40- 50 was ensured throughout the procedure. Sevoflurane or any other volatile anesthetic agent was not utilized in Group-P at any time during procedure.

In both groups during pre-CPB period, any hypotension was treated with phenylephrine boluses and hypertension with nitroglycerin infusion to a target MAP of > 65mmHg, respectively. Patients requiring repeated boluses of phenylephrine were treated with norepinephrine infusion (0.05 - 0.1 mcg/kg/min).

Volume control ventilation mode was used for ventilation during procedure. Heart rate, 5 - lead electrocardiography with ST analysis, invasive blood pressure, pulse oximetry, respiratory gas monitoring, central venous pressure, BIS and body temperature were monitored in both groups during procedure. AVR with CPB using heart lung machine (Jostra HL20, Maquet, Rastatt, Germany) with non-pulsatile perfusion using roller pump

and membrane type oxygenators with an appropriate flow for the core temperature was performed in all patients. Intermittent antegrade cardioplegia with cold blood followed by cardioplegia administration through coronary ostia post aortotomy was done in all patients. This study utilized cases operated specifically by two surgeons as first operators.

During CPB weaning, phenylephrine boluses of 1 - 2 mcg/kg were injected if MAP was less than 65 mm Hg. Norepinephrine was started at a dose of 0.05 mcg/kg/min if desired cardiac index and MAP were not achieved despite repeated phenylephrine boluses. Use of inotropes like epinephrine and dobutamine were restricted to only those cases wherein poor LV contractility was demonstrated by TEE.

Patients were transferred to the ICU at the end of procedure. An infusion of 20 mcg/kg/hr of morphine was used for ICU sedation and analgesia. Patients were weaned from the ventilator and trachea was extubated once they were warm with stable hemodynamic parameters.

2D STE protocol

Pre-CPB STE protocol:

Philips ultrasound workstation (iE33) with RT-3D-TEE probes was used for echocardiography in all patients. All 2D echocardiographic images were acquired at stable hemodynamics (± 20% of the basal values for the CVP, heart rate, MAP and left ventricular end diastolic area). 2D echocardiography as per ASE guidelines, was utilized to measure ejection fraction, stroke volume, cardiac output, LV thickness, LV mass, and E/E'. Global longitudinal strain was calculated using 2D echocardiographic images acquired from mid-esophageal (ME) 4-chamber view, 2-chamber view and 3-chamber view (long-axis view) with optimal gain and frame rate more than 50 per second. Transgastric basal, mid and apical views with optimal gain and frame rate more than 50 per second were utilized to calculate global circumferential strain.

Post-CPB STE protocol:

2D echocardiographic and doppler calculation of prosthetic valve peak velocity, prosthetic valve mean gradient, acceleration time, effective orifice area index, doppler velocity index, paravalvular leak, cardiac output, TAPSE and SVR were done. These were done post-sternal closure, while patient was in sinus rhythm and without inotropes in majority of patients. All 2D echocardiographic images were acquired at stable hemodynamics (± 20% of the basal values for the CVP, heart rate, MAP and left ventricular end diastolic area. Global longitudinal strain was calculated using 2D echocardiographic images acquired from mid-esophageal (ME) 4-chamber view, 2-chamber view and 3-chamber view (long-axis view) with optimal gain and frame rate more than 50 per second. Transgastric basal, mid and apical views with optimal gain and frame rate more than 50 per second were utilized to calculate global circumferential strain.

2D echocardiographic parameters analysis:

Ejection fraction, stroke volume, cardiac output, LV mass, LV thickness and E/E' were calculated on Philips iE33 system in operation theatre.

Analysis of longitudinal and Circumferential strain:

Echocardiographic loops as described by EACVI/ASE acquired at midesophageal (ME) 4Ch, ME 2Ch, ME 3Ch (LAX), Transgastric (TG) basal, TG mid papillary and TG apical views with similar heart rate (within 10 beats/ min) were used to calculate GLS and GCS. Electrocardiogram from a single lead (lead I/II) was recorded in loops to aid in GLS and GCS calculation.

A frame rate of more than 50 frames per second was achieved in all loops using sector width and depth adjustment. Echocardiographic images that clearly visualized endocardial and epicardial borders with least artifacts were selected for analysis. Loops comprising of three cardiac cycles were acquired for each imaging sector and was stored as cine-loops in DICOM format.

Experienced cardiac anesthesiologists performed offline strain analysis using a specialized software installed on Philips iE33 (QLAB 9.1). GLS and GCS were derived by this software using semi-automatic detection and demarcation of endocardial and epicardial borders followed by cardiac motion quantification (tracking of ultrasonic speckles in a cardiac cycle).

The technique used for analysis of GLS was a three-click marker method, wherein the evaluator placed three markers at each side of mitral annulus and LV apex for ME 4Ch and ME 2Ch view. In ME 3Ch (LAX) view, the markers were placed at posterior mitral annulus, topmost point of basal anterior septal wall and LV apex.

For calculation of GCS, the software automatically tracked epicardial and endocardial borders of transgastric views, which could further be adjusted by operator using track ball movement.

The duration of aortic valve closure from end diastole was automatically evaluated by software. This was reconfirmed by the operator manually. In addition, the operator also visually assessed the quality of tracking of region of interest during playback of echocardiography loop. The width of the region of interest was also corrected to the epicardial and endocardial borders if needed. Operator also rejected loops with inadequate tracking and those patients were subsequently excluded from study.

A software created color-coded parametric image in bull's eye display presented the global longitudinal and circumferential strain together with concurrent segmental strain values for quick visual analysis and evaluation.

All strain values were described in negative percentage numbers, so that an increase in longitudinal and circumferential strain showed a higher negative percentage number when compared to a decrease in longitudinal and circumferential strain, that depicted a lower number.

Manual entry of study data was done in the study proforma as attached in the annexure

Postoperative parameters

Vasoactive-inotropic score (45) was used to calculate postoperative inotropic requirement. We also documented other parameters like time to extubation from arrival in ICU, duration of ICU stay and post operative transthoracic echocardiography derived peak prosthetic valve velocity with mean gradient and EF.

Figure 4: Calculation of VIS

Inotropic Score (IS) = dopamine dose (μg/kg/min)
+ dobutamine dose (μg/kg/min)
+ 100 × epinephrine dose (μg/kg/min)

Vasoactive-Inotropic Score (VIS) =

IS + 10 × PDE inhibitor (milrinone or olprinone) dose (μg/kg/min)
+ 100 × norepinephrine dose (μg/kg/min)
+ 10000 × vasopressin dose (U/kg/min)

STATISTICAL ANALYSIS

STATISTICAL ANALYSIS

Pre-CPB and Post-CPB GLS with Pre-CPB and Post-CPB GCS were considered as primary outcome variables. Study groups were considered as secondary outcome variables. VIS score was considered as primary explanatory variable.

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Variables, which were not distributed normally were summarized by median and interquartile range (IQR). Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots.

All quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of > 0.05 was considered as normal distribution.

Categorical outcomes were compared between study groups using Chi square test /Fisher's exact test (If the overall sample size was < 20 or if the expected number in any one of the cells is < 5, Fisher's exact test was used.)
For normally distributed quantitative parameters the mean values were compared between study groups using independent sample t-test (2 groups). The change in the quantitative parameters, before and after the intervention was assessed by paired t-test (In case of two time periods).

Association between quantitative explanatory and outcome variables was assessed by calculating Pearson correlation coefficient and the data was represented in a scatter diagram. Intra and inter class correlation was assessed using intra/interclass correlation coefficient.

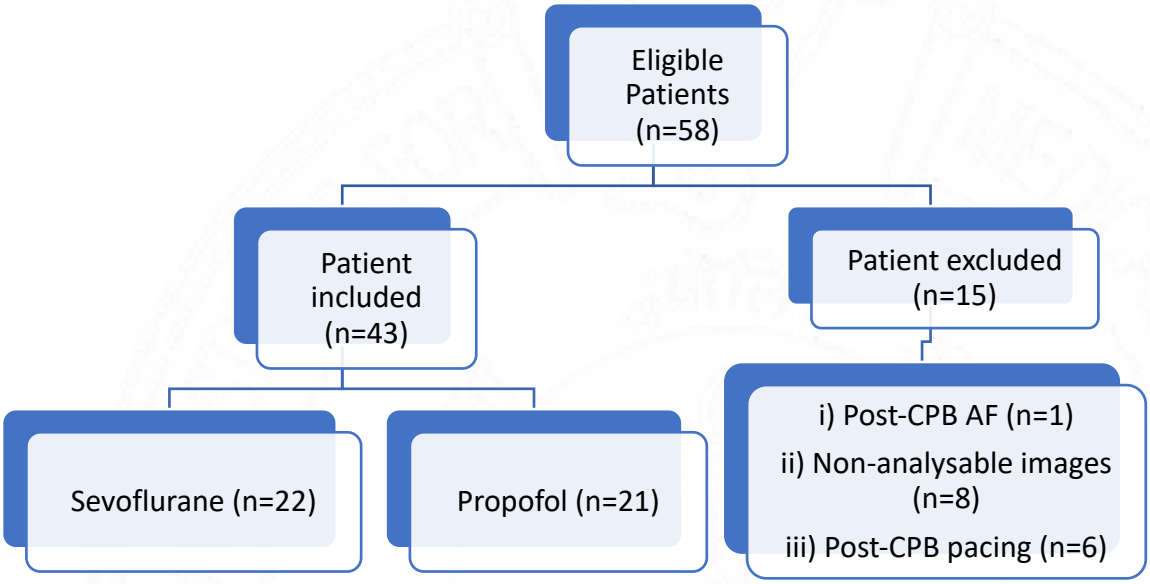
P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.

RESULTS

OBSERVATION AND RESULTS

Results: A total of 43 subjects were included in the final analysis.

Figure 5: Consort Diagram For patient selection



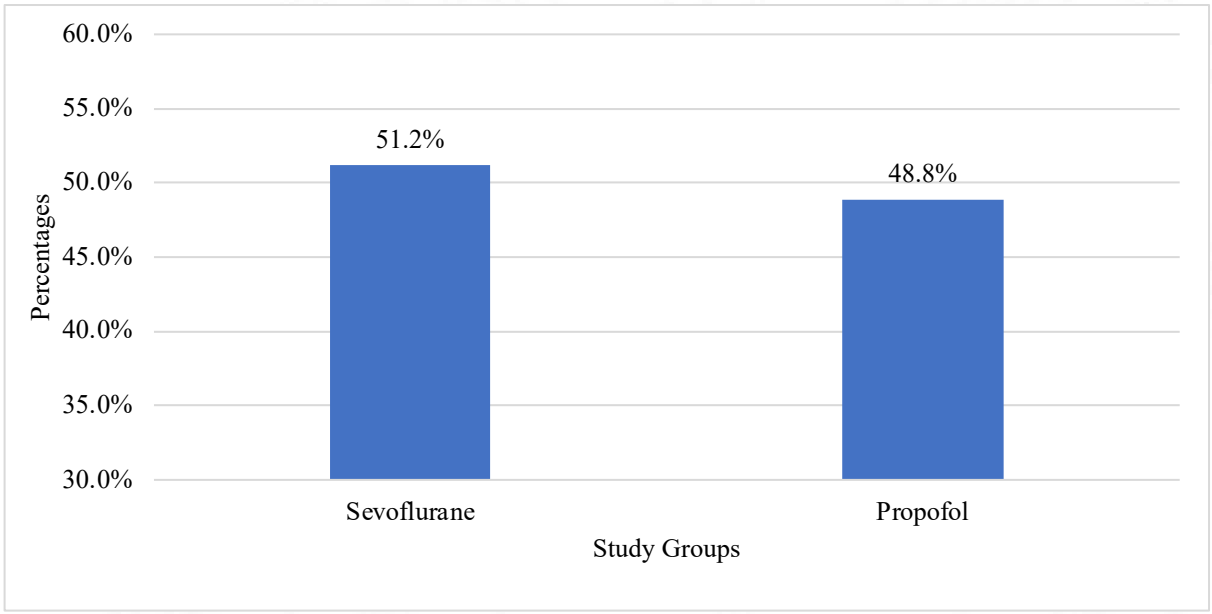
Consort diagram explaining patient selection. 58 patients were considered eligible; 15 were excluded because of post-CPB atrial fibrillation (n=1), non-analyzable images (n=8) and post-CPB pacing requirement (n =6). Forty-three patients were included, who were distributed between Sevoflurane group (n=22) and Propofol group (n=21).

Table 1: Descriptive analysis of study group in the study population (n=43)

Study Group	Frequency	Percentages
Sevoflurane	22	51.16%
Propofol	21	48.84%

Table 1 depicts frequency and percentage distribution of patients in Sevoflurane and Propofol group.

Figure 6: Bar chart of group in the study population (n=43)



Bar chart describes percentage of patients in Sevoflurane and Propofol groups

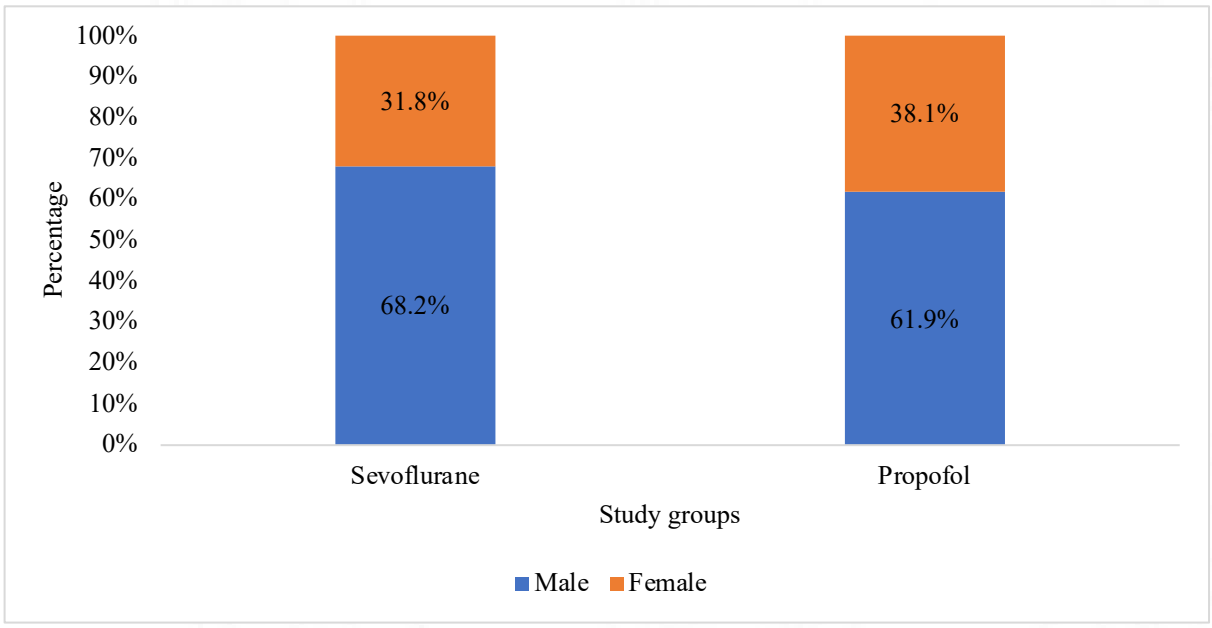
Table 2: Comparison of demographic parameter between study groups (n=43)

Demographic parameter	Study Groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	**Propofol (n=21)**	
Age (years)	61.5 ± 8.86	61.62 ± 3.96	0.955
BSA (m^2)	1.74 ± 0.24	1.67 ± 0.17	0.241
Gender			
Male	15 (68.18%)	13 (61.9%)	0.666
Female	7 (31.82%)	8 (38.1%)	

On comparing the demographic parameters between the study groups, no significant difference was observed in terms of age, body surface area and gender.

Abbreviations: - BSA: Body surface area

Figure 7: Staked bar chart of comparison of gender between study groups (n=43)



Staked bar chart shows no difference in gender between the groups.

Table 3: Comparison of risk factors between study groups (n=43)

Risk factors	Study group		Chi square	*p - value*
	Sevoflurane (n=22)	Propofol (n=21)		
Hypertension				
Yes	13 (59.09%)	8 (38.1%)	1.896	0.169
No	9 (40.91%)	13 (61.9%)		
Diabetes Mellitus				
Yes	10 (45.45%)	8 (38.1%)	0.239	0.625
No	12 (54.55%)	13 (61.9%)		
CCB Use				
Yes	5 (22.73%)	5 (23.81%)	0.007	1.000*
No	17 (77.27%)	16 (76.19%)		
ARB/ ACEI Use				
Yes	6 (27.27%)	4 (19.05%)	0.407	0.721*
No	16 (72.73%)	17 (80.95%)		
Beta Blocker Use				
Yes	6 (27.27%)	2 (9.52%)	2.235	0.240*
No	16 (72.73%)	19 (90.48%)		

Note: * Fishers exact p-value. Fishers exact test was used to ascertain statistical significance between the groups as the overall sample size was less than 20 and the sample size in any one of the cells was less than 5.

Table 3 depicts that there was no difference between the groups for possible risk factors influencing the strain values such as hypertension, diabetes mellitus, CCB use, ARB/ACEI use and beta blocker use.

Abbreviations: - CCB: Calcium channel blocker; ARB: Angiotensin receptor antagonist; ACEI: Angiotensin converting enzyme inhibitor.

Table 4: Comparison of mean of preoperative 2D transthoracic echocardiographic and doppler parameters between the study groups (n=43)

Preoperative Details	Study Groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	Propofol (n=21)	
Septal Wall Thickness Systole (mm)	17.09 ± 4.05	15.38 ± 3.83	0.163
Septal Wall Thickness Diastole (mm)	14.18 ± 3.47	12.33 ± 3.18	0.077
Inferolateral Wall Thickness Systole (mm)	15.86 ± 3.47	14.76 ± 3.48	0.304
Inferolateral Wall Thickness Diastole (mm)	13.09 ± 3.24	11.38 ± 2.46	0.059
EF (%)	63 ± 9.3	66.05 ± 5.83	0.208
Aortic Valve Mean Gradient (mmHg)	54.14 ± 12.16	53.86 ± 11.52	0.939
Aortic Valve Peak Velocity (metre/second)	4.55 ± 0.55	4.52 ± 0.57	0.878

Table 4 shows that no statistically significant differences were observed on comparison of mean of transthoracic echocardiographic and doppler parameters such as septal wall thickness in systole, septal wall thickness in diastole, inferolateral wall thickness in systole, inferolateral wall thickness in diastole, ejection fraction, aortic valve mean gradient and aortic valve peak velocity between the study groups.

Average ejection fraction was within normal limits. Doppler interrogation reveals severe AS.

Abbreviation: - EF: Ejection fraction

Table 5: Comparison of mean of hemodynamic, 2D tranosesophageal and doppler echocardiographic parameters in the pre-CPB period between study groups (n=43)

Pre-CPB data	Study groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	Propofol (n=21)	
Pre-CPB heart rate	70.14 ± 15.99	71.71 ± 10.26	0.704
Systolic BP	124.36 ± 15.96	124.9 ± 14.55	0.908
Diastolic BP	69.41 ± 11.64	72.62 ± 8.59	0.311
Mean arterial pressure	87.05 ± 11.32	86.52 ± 15.36	0.899
Pulse pressure	54.95 ± 12.94	53.86 ± 17.32	0.815
CVP	9.45 ± 1.99	8.81 ± 2.44	0.347
LVEDA (cm^2)	15.51 ± 8.62	13.71 ± 2.17	0.359
LVOT CSA (cm^2)	3.4 ± 0.54	3.3 ± 0.39	0.521
LVOT VTI (cm)	17.24 ± 2.86	18.73 ± 2.42	0.073
LVOT SV (ml)	59.14 ± 16.66	60.87 ± 6.9	0.662
LVOT SV index (ml/m^2)	33.97 ± 9.15	36.72 ± 4.6	0.224
LVOT CO (L/min)	3.94 ± 0.91	4.32 ± 0.75	0.143
Aortic valve mean gradient (mmHg) under GA	43.36 ± 8	39.81 ± 9.36	0.187
SVR ($dynes/sec/cm^{-5}$)	1624.09 ± 487.45	1552.29 ± 400.34	0.601
Systemic arterial compliance	0.64 ± 0.23	0.8 ± 0.31	0.075
Valvuloarterial impedance	5.21 ± 1.62	4.29 ± 0.98	0.051
Septal wall thickness (mm)	14.48 ± 2.81	13.2 ± 1.88	0.088
Inferolateral wall thickness (mm)	14.06 ± 2.78	13.15 ± 2.21	0.240
Left ventricular internal diameter (mm)	38.36 ± 5.24	40.56 ± 4.13	0.135
LV long axis Length (mm)	83.65 ± 8.28	82.47 ± 19.58	0.797
LV Mass (gm)	245.59 ± 54.31	232.71 ± 27.56	0.336
LV Mass Index (gm/m^2)	142.3 ± 33.75	142 ± 24.14	0.974
E/E'	10.14 ± 3.08	11.8 ± 3.27	0.094
Relative wall thickness	0.72 ± 0.16	0.64 ± 0.13	0.103

Table 5 comparing hemodynamic, doppler and 2D transoesophageal echocardiographic parameters in pre-CPB period between study groups shows significant p- value difference only in systemic arterial compliance.

Abbreviations: - CPB: Cardiopulmonary bypass; BP: Blood pressure; CVP: Central venous pressure; LVEDA: Left ventricular end diastolic area; LVOT: Left ventricular outflow tract; CSA: cross sectional area; VTI: Velocity time integral; SV: Stroke volume; CO: cardiac output; SVR: Systemic vascular resistance; LV: Left ventricular.

Table 6: Comparison of pre-CPB GLS parameters; mean of midoesophageal 2Ch, 3Ch and 4Ch regional strains between study groups (n = 43)

Pre-CPB GLS parameters	Study groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	Propofol (n=21)	
ME 2Ch strain			
Basal Anterior	-8.95 ± 4.59	-13.14 ± 5.41	0.009*
Mid Anterior	-10.5 ± 5.6	-12.86 ± 3.76	0.114
Apical Anterior	-13.73 ± 7.94	-16.1 ± 7.82	0.330
Basal Inferior	-11.27 ± 7.42	-13.76 ± 4.17	0.185
Mid Inferior	-11.32 ± 4.45	-12.57 ± 6.61	0.468
Apical Inferior	-13.64 ± 6.53	-16.86 ± 7.01	0.126
Apex	-12.86 ± 7.05	-17.29 ± 6.69	0.041*
ME 3Ch strain (LAX)			
Basal Anteroseptal	-8.73 ± 4.76	-8.24 ± 7.78	0.804
Mid Anteroseptal	-9.41 ± 6.64	-12.71 ± 7.06	0.121
Apical Anteroseptal	-11.32 ± 4.99	-12.24 ± 6.03	0.588
Basal Inferolateral	-10.14 ± 6.08	-10.81 ± 6.56	0.729
Mid Inferolateral	-9.68 ± 5.54	-11.24 ± 2.86	0.257
Apical Inferolateral	-11.5 ± 4.11	-11.38 ± 5.91	0.939
Apex	-11.59 ± 4.39	-11.71 ± 4	0.924
ME 4Ch strain			
Basal Anterolateral	-9.91 ± 3.45	-11.43 ± 5.05	0.254
Mid Anterolateral	-9 ± 6.98	-11.86 ± 5.89	0.156
Apical Anterolateral	-11.91 ± 5.82	-13.67 ± 4.7	0.284
Basal Inferoseptal	-10.91 ± 4.47	-11.52 ± 3.06	0.603
Mid Inferoseptal	-8.14 ± 7.64	-13.48 ± 4.97	0.010*
Apical Inferoseptal	-10.36 ± 6.67	-14.1 ± 6.11	0.063
Apex	-10.5 ± 5	-11.81 ± 5.5	0.418

Table 6 comparing pre-CPB GLS parameters between groups showing a significant p-value difference in ME 2Ch basal anterior, ME 2Ch Apex and ME 4Ch mid inferoseptal regions. Overall segmental strains were more negative in apical area compared to corresponding basal area of given LV wall.

Abbreviations: - GLS: Global longitudinal strain; ME 2Ch: Mid oesophageal 2 chamber, ME 3Ch: Mid oesophageal 3 chamber, ME 4Ch: Mid oesophageal 4 chamber; LAX: Long axis

* indicates statistically significant values.

Table 7: Comparison of pre-CPB GCS parameters; mean of transgastric basal, mid & apical regional strains between study groups (n=43)

pre-CPB GCS parameters	Study groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	Propofol (n=21)	
TG Basal Strain			
Inferior	-24 ± 6.35	-23.1 ± 6.49	0.647
Inferoseptal	-22.23 ± 10.68	-18.95 ± 11.05	0.329
Anteroseptal	-23.59 ± 9.63	-23.67 ± 5.25	0.975
Anterior	-28.32 ± 7.49	-25.9 ± 6.82	0.276
Anteriolateral	-27.27 ± 5.82	-24 ± 6.37	0.086
Inferolateral	-24.36 ± 5.94	-22 ± 6.27	0.211
TG Mid Strain	-27.14 ± 5.53	-24.86 ± 4.96	0.163
Inferior	-27.41 ± 9.62	-25.95 ± 6.34	0.563
Inferoseptal	-25.68 ± 6.43	-23.33 ± 8.52	0.312
Anteroseptal	-28.14 ± 9.82	-23.29 ± 9.72	0.111
Anterior	-32.59 ± 6.09	-28.1 ± 7.31	0.034*
Anterolateral	-26.41 ± 7.93	-24.38 ± 5.87	0.348
Inferolateral	-26.86 ± 6.93	-25.71 ± 6.61	0.581
TG Apical Strain	-26 ± 6.61	-27.14 ± 4.45	0.512
Inferior	-26.36 ± 8.03	-28.71 ± 6.27	0.292
Septal	-26.68 ± 7.64	-27.57 ± 8.65	0.722
Anterior	-27 ± 8.54	-26.95 ± 7.28	0.984
Lateral	-24.95 ± 6.93	-25.95 ± 7.89	0.661

Table 7 comparing pre- CPB GCS parameters between groups depicting no statistically significant difference between sevoflurane and propofol group other than in TG mid anterior region.

There was no consistency in the difference among basal and apical segmental circumferential strain, as observed in the longitudinal strain.

Abbreviations: - GCS: Global circumferential strain; TG: Transgastric

* indicates statistically significant values.

Table 8: Comparison of mean of pre-CPB GLS and GCS between risk factors (n=43)

Risk factor	(Mean± SD)		*p - value*
Hypertension	**Yes (n=21)**	**No (n=22)**	
Pre-CPB GLS	-11 ± 2.57	-11.86 ± 2.64	0.284
Pre-CPB GCS	-26.24 ± 3.4	-24.36 ± 3.76	0.095
Diabetes Mellitus	**Yes (n=18)**	**No (n=25)**	
Pre-CPB GLS	-11.33 ± 2.54	-11.52 ± 2.71	0.820
Pre-CPB GCS	-25.5 ± 3.5	-25.12 ± 3.85	0.742
CCB use	**Yes (n=10)**	**No (n=33)**	
Pre-CPB GLS	-11.3 ± 2.71	-11.48 ± 2.62	0.847
Pre-CPB GCS	-27.4 ± 2.32	-24.64 ± 3.79	0.035*
ARB/ ACEI Use	**Yes (n=10)**	**No (n=33)**	
Pre-CPB GLS	-9.8 ± 1.75	-11.94 ± 2.65	0.021*
Pre-CPB GCS	-25.6 ± 4.2	-25.18 ± 3.57	0.757
Beta Blocker Use	**Yes (n=8)**	**No (n=35)**	
Pre-CPB GLS	-10.75 ± 2.76	-11.6 ± 2.59	0.413
Pre-CPB GCS	-26.13 ± 3.18	-25.09 ± 3.79	0.477

Table 8 contrasting mean of pre-CPB GLS and GCS between various risk factors. Patients using CCB were found to having statistically significant higher pre-CPB GCS score when compared to those patients not using CCB.

In addition, patients on ARB/ACE inhibitor were found to be having a lower pre-CPB GLS score when compared to those not on them.

Abbreviations: - CCB: Calcium channel blocker; ARB: Angiotensin receptor antagonist; ACEI: Angiotensin converting enzyme inhibitor

* indicates statistically significant values.

Table 9: Comparison of mean of CPB and valve details between study groups (n = 43)

Parameter	Study Groups (Mean± SD)		*p- value*
	Sevoflurane (n=22)	**Propofol (n=21)**	
CPB details			
Clamp Time (min)	98.14 ± 26.89	83.62 ± 33.34	0.123
CPB Time (min)	148.18 ± 40.32	118.33 ± 44.48	0.026*
Valve Details			
Valve Size	21.09 ± 2	20.62 ± 1.5	0.388

Table 9 comparing CPB details and valve details between study groups shows a statistically significant difference in mean CPB time between both groups; sevoflurane group CPB time being more than propofol group which may be attributed to surgeries performed by two different surgical teams.

There is no difference in clamp time or valve size between both groups.

* indicates statistically significant values.

Table 10: Comparison of valve type between study groups (n=43)

Valve Type	Study Groups	
	Sevoflurane (N=22)	**Propofol (N=21)**
TTK Chitra (Tilting-disc valve)	12 (54.55%)	11 (52.38%)
Inspiris Resilia (Stented bovine pericardial valve)	1 (4.55%)	0 (0%)
ON – X (Bi-leaflet mechanical valve)	2 (9.09%)	2 (9.52%)
Perimount Magna (Stented bioprosthetic valve)	7 (31.82%)	8 (38.1%)

Table 10 showing types of various valves implanted in different study group

Figure 8: Cluster bar chart of comparison of valve type between study groups (n=43)

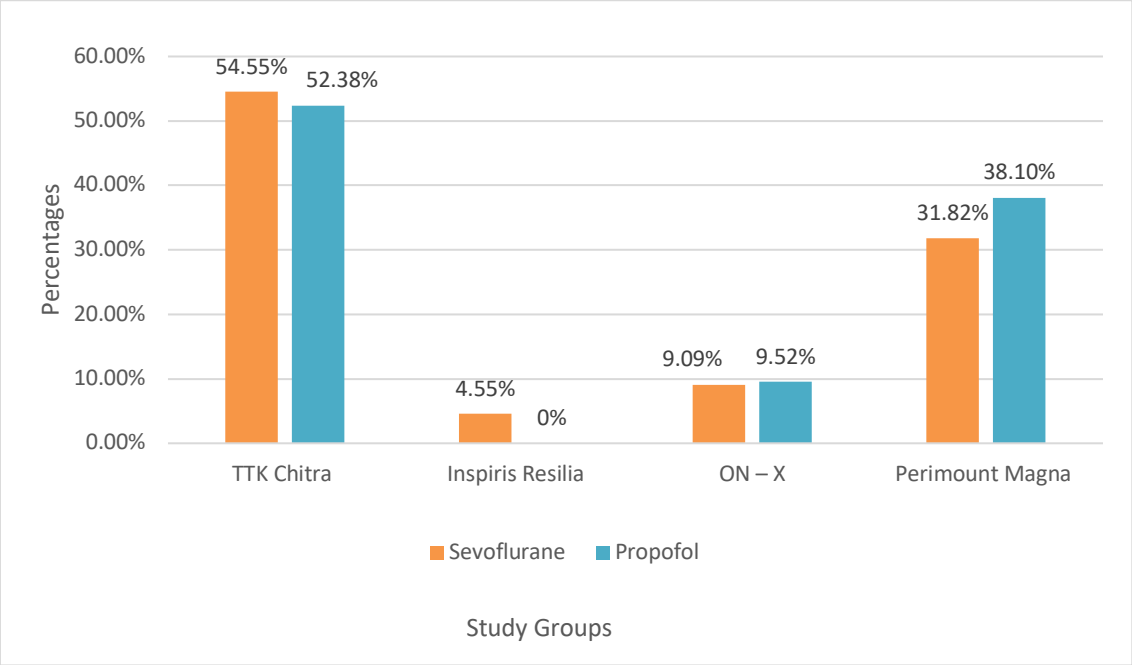


Figure 8 depicting distribution of various valves implanted in different study groups

Table 11: Comparison VIS on weaning from CPB between study groups(n=43)

Parameter	Study Groups (Mean± SD)		P value
	Sevoflurane (N=22)	Propofol (N=21)	
VIS while weaning from CPB	4.5 ± 3.55	3.71 ± 4.03	0.501

Table 11 showing similar VIS between sevoflurane and propofol groups while weaning from CPB

Abbreviations: - VIS: Vasopressor inotrope score

Table 12: Comparison of mean of post-CPB parameters between study groups(n=43)

Post-CPB Parameter	Study groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	Propofol (n=21)	
Post-CPB Hb (mg/dl)	9.42 ± 0.92	10.2 ± 0.72	0.004*
LVEDA (cm^2)	13.67 ± 3.07	12.57 ± 1.78	0.160
Heart rate (per minute)	74.45 ± 11.25	75 ± 11.51	0.876
Aortic valve prosthesis peak Velocity (metre/second)	2.24 ± 0.66	2.07 ± 0.49	0.353
Aortic valve prosthesis mean gradient (mmHg)	11.41 ± 4.25	11.24 ± 3.97	0.892
Acceleration Time (milli second)	77.86 ± 8.92	73.57 ± 12.54	0.201
EOAI (cm^2/m^2)	0.91 ± 0.33	0.89 ± 0.23	0.817
DVI	0.49 ± 0.22	0.47 ± 0.13	0.733
LVOT Cardiac Output (Litre/min)	3.7 ± 0.84	3.5 ± 1.01	0.505
TAPSE (mm)	18.45 ± 2.11	18.57 ± 1.72	0.844
SVR (dynes/sec/cm^{-5})	1150.73 ± 218.49	1228.95 ± 225.31	0.254

Table 12 comparing post-CPB parameters between study groups showing a statistically significant difference between groups in post-CPB hemoglobin; propofol group having a higher value when compared to sevoflurane group.

Abbreviations: - Hb: Hemoglobin; LVEDA: Left ventricular end diastolic area; EOAI: Indexed effective orifice area; DVI: Doppler velocity index; LVOT: Left ventricular outflow tract; TAPSE: Tricuspid annular plane systolic excursion.

* indicates statistically significant values.

Table 13: Comparison of post-CPB GLS parameters; mean of midoesophageal 2Ch, 3Ch and 4Ch strains between study groups (n = 43)

Post-CPB GLS parameters	Study groups (Mean± SD)		*p - value*
	Sevoflurane (n=22)	Propofol (n=21)	
ME 2Ch strain			
Basal Anterior	-8.86 ± 5.83	-10.52 ± 6.74	0.392
Mid Anterior	-10.45 ± 7.02	-11.52 ± 4.66	0.561
Apical Anterior	-12.14 ± 8.55	-14.67 ± 8.38	0.333
Basal Inferior	-9.68 ± 8.33	-13.1 ± 6.05	0.134
Mid Inferior	-9.64 ± 10.94	-12.24 ± 4.12	0.313
Apical Inferior	-11.27 ± 4.79	-12.52 ± 6.57	0.478
Apex	-11.09 ± 6.6	-13 ± 7.42	0.377
ME 3Ch strain (LAX)			
Basal Anteroseptal	-6.68 ± 9.76	-11.38 ± 6.1	0.067
Mid Anteroseptal	-9.95 ± 8.48	-11.95 ± 6.08	0.382
Apical Anteroseptal	-13.45 ± 7.94	-13.43 ± 4.82	0.990
Basal Inferolateral	-10.09 ± 6.91	-11.52 ± 5.67	0.463
Mid Inferolateral	-11.91 ± 5.25	-9.67 ± 7.72	0.270
Apical Inferolateral	-10.27 ± 6.97	-12.43 ± 3.16	0.202
Apex	-11.41 ± 6.29	-12.52 ± 4.34	0.505
ME 4Ch strain			
Basal Anterolateral	-10.27 ± 6.36	-12.71 ± 5.82	0.197
Mid Anterolateral	-11.41 ± 5.96	-12.29 ± 4.65	0.595
Apical Anterolateral	-12.55 ± 4.44	-13.9 ± 6.48	0.425
Basal Inferoseptal	-11.09 ± 3.64	-12.48 ± 3.88	0.234
Mid Inferoseptal	-10.09 ± 6.77	-12.62 ± 4.26	0.153
Apical Inferoseptal	-11.59 ± 5.6	-13.05 ± 5.06	0.377
Apex	-10.55 ± 4.89	-12.62 ± 6.01	0.221

Table 13 comparing post-CPB GLS parameters between both groups depicting no statistically significant difference.

Table 14: Comparison of Post-CPB GCS parameters; mean of transgastric basal, mid & apical strains between study groups (n=43)

Post-CPB GCS parameters	Study groups (Mean± SD)		*p - value*
	Sevoflurane (N=22)	**Propofol (N=21)**	
TG Basal Strain	-22.23 ± 3.83	-21.52 ± 2.71	0.493
Inferior	-22.05 ± 6.46	-22.48 ± 6	0.822
Inferoseptal	-22.64 ± 5.21	-21.67 ± 5.17	0.544
Anteroseptal	-22.86 ± 4.87	-20.81 ± 8.51	0.334
Anterior	-21.59 ± 9.11	-23.62 ± 6.53	0.408
Anteriolateral	-21.91 ± 5.07	-21.05 ± 5.3	0.589
Inferolateral	-22.36 ± 7.36	-20.76 ± 9.14	0.529
TG Mid Strain	-23.55 ± 4.86	-24.81 ± 5.43	0.425
Inferior	-24.05 ± 6.68	-25.33 ± 6.88	0.537
Inferoseptal	-22.73 ± 7.67	-24.67 ± 7.21	0.398
Anteroseptal	-25.09 ± 6.55	-26 ± 7.58	0.675
Anterior	-23.95 ± 6.45	-25.81 ± 8.13	0.411
Anterolateral	-24.32 ± 6.08	-22.81 ± 9.26	0.529
Inferolateral	-23.18 ± 6.22	-24.19 ± 6.05	0.593
TG Apical Strain	-23.36 ± 5.28	-22.29 ± 6.28	0.545
Inferior	-24.32 ± 6.61	-24.05 ± 7.39	0.900
Septal	-22.55 ± 5.98	-23.95 ± 8.03	0.517
Anterior	-23.55 ± 7.91	-22.19 ± 7.85	0.576
Lateral	-23.77 ± 5	-18.52 ± 10.3	0.038*

Table 14 comparing post-CPB GCS parameters between both groups depicting no statistically significant difference other than TG apical region.

* indicates statistically significant values.

Table 15: Comparison of mean of post operative details between study groups(n=43)

Post Op Details	Study Groups (Mean± SD)		*p - value*
	Sevoflurane (N=22)	**Propofol (N=21)**	
VIS on arrival in ICU	4.64 ± 5.2	1.6 ± 2.22	0.018*
Duration of ventilation (hours)	14.05 ± 3.81	12.48 ± 3.14	0.149
ICU stay (hours)	40.09 ± 12.55	47.14 ± 14.61	0.097
Peak velocity (metre/second)	1.84 ± 0.51	2.13 ± 0.58	0.089
Mean Gradient (mm Hg)	10.36 ± 3.9	11.81 ± 4.25	0.251
EF (%)	61.59 ± 8.67	64.67 ± 6.89	0.206

Table 15 comparing VIS score at arrival in ICU showing sevoflurane having more VIS compared to propofol that is statistically significant

Abbreviations: - VIS: Vasopressor inotrope score; ICU: Intensive care unit

* indicates statistically significant values.

Table 16: Comparison of mean of pre-CPB and post-CPB GLS, GCS and regional parameters between study groups (n=43)

Parameter	Study Groups (Mean ± SD)		*p - value*
	Sevoflurane (n=22)	**Propofol (n=21)**	
Pre-CPB GLS	-10.41 ± 2.46	-12.52 ± 2.36	0.003*
Pre ME 2Ch strain	-11.36 ± 4.18	-14.48 ± 4.64	0.013*
Pre ME 3Ch strain (LAX)	-10.23 ± 2.27	-11.1 ± 2.98	0.144
Pre ME 4Ch strain	-9.91 ± 2.83	-12.43 ± 2.99	0.004*
Pre-CPB GCS	-26.18 ± 3.97	-24.33 ± 3.15	0.050
Pre TG basal strain	-25.09 ± 4.35	-22.76 ± 3.99	0.037*
Pre TG mid strain	-27.14 ± 5.53	-24.86 ± 4.96	0.082
Pre TG apical strain	-26 ± 6.61	-27.14 ± 4.45	0.256
Post-CPB GLS	-10.68 ± 3.09	-11.9 ± 2.49	0.081
Post ME 2Ch Strain	-10.5 ± 4.87	-12.1 ± 3.91	0.122
Post ME 3Ch Strain (LAX)	-10.59 ± 4.2	-11.48 ± 3.11	0.219
Post ME 4Ch Strain	-10.86 ± 3.51	-12.62 ± 3.87	0.063
Post-CPB GCS	-22.95 ± 3.97	-23 ± 4.16	0.485
Post TG basal Strain	-22.23 ± 3.83	-21.52 ± 2.71	0.246
Post TG mid Strain	-23.55 ± 4.86	-24.81 ± 5.43	0.213
Post TG apical Strain	-23.36 ± 5.28	-22.29 ± 6.28	0.272

Table 16 showing comparison of strain parameters between study groups in pre-CPB and post-CPB period. There is a statistically significant difference in pre-CPB GLS with pre-CPB ME 2Ch and ME 4Ch strain between groups, which was found to be higher in propofol group.

A statistically significant difference was also observed in pre-CPB transgastric strain between groups in basal region. No other intergroup difference was observed in pre-CPB and post-CPB GLS and GCS values.

* indicates statistically significant values.

Table 17: Comparison of mean of GLS and related parameters between pre-CPB and post-CPB follow up time periods (n=43)

Follow-up periods	(Mean± STD)	Mean Difference	95% CI of mean difference		*p - value*
			Lower	Upper	
GLS					
Pre-CPB GLS	-11.44 ± 2.61	0.16	0.49	0.82	0.620
Post-CPB GLS	-11.28 ± 2.85				
ME 2Ch Strain					
Pre ME 2Ch strain	-12.88 ± 4.64	1.60	0.30	2.91	0.017*
Post ME 2Ch strain	-11.28 ± 4.45				
ME 3Ch Strain (LAX)					
Pre ME 3Ch strain	-10.65 ± 2.64	0.37	0.75	1.49	0.506
Post ME 3Ch strain	-11.02 ± 3.69				
ME 4Ch Strain					
Pre ME 4Ch strain	-11.14 ± 3.14	0.58	0.67	1.83	0.355
Post ME 4Ch strain	-11.72 ± 3.75				

Table 17 comparing mean of GLS and related parameters during pre-CPB and post-CPB period shows a statistically significant decrease in midoesophageal two chamber strain in post-CPB period when compared to pre-CPB period. There was no other difference in pre-CPB and post-CPB period in GLS or any of its other regional parameters.

* indicates statistically significant values.

Table 18: Comparison of mean of GCS and related parameters between pre-CPB and post-CPB follow up time periods (n=43)

Follow-up periods	(Mean± STD)	Mean Difference	95% CI of mean difference		*p - value*
			Lower	Upper	
GCS					
Pre-CPB GCS	-25.28 ± 3.67	2.30	1.06	3.54	<0.001*
Post-CPB GCS	-22.98 ± 4.01				
TG Basal Strain					
Pre TG basal Strain	-23.95 ± 4.29	2.07	1.15	2.99	<0.001*
Post TG basal Strain	-21.88 ± 3.31				
TG Mid Strain					
Pre TG mid Strain	-26.02 ± 5.33	1.86	0.01	3.71	0.049*
Post TG mid Strain	-24.16 ± 5.12				
TG Apical Strain					
Pre TG apical Strain	-26.56 ± 5.62	3.72	1.49	5.95	0.002*
Post TG apical Strain	-22.84 ± 5.74				

Table 18 comparing mean of GCS and related parameters during pre-CPB and post-CPB period shows a statistically significant decrease in GCS and all its regional values in post CPB.

* indicates statistically significant values.

Table 19: Comparison of preoperative and postoperative EF (n=43)

Follow-up periods	(Mean± STD)	Mean Difference	95% CI of mean difference		P-value
			Lower	Upper	
Preoperative EF (%)	64.49 ± 7.86	1.40	1.65	4.44	0.361
Postoperative EF (%)	63.09 ± 7.91				

Table 19 shows comparison between EF, that shows no difference between preoperative and postoperative period

Table 20: Comparison of mean of GLS and regional parameters between pre-CPB and post -CPB follow up time periods (within study group) (n=43)

Follow-up periods	(Mean± STD)	Mean Difference	95% CI of mean difference		*p-value*
			Lower	Upper	
Sevoflurane (n=22)					
GLS					
Pre-CPB GLS	-10.41 ± 2.46	0.27	0.81	1.35	0.605
Post-CPB GLS	-10.68 ± 3.09				
ME 2Ch strain					
Pre ME 2Ch strain	-11.36 ± 4.18	0.86	1.26	2.98	0.407
Post ME 2Ch strain	-10.5 ± 4.87				
ME 3Ch strain (LAX)					
Pre ME 3Ch strain	-10.23 ± 2.27	0.36	1.62	2.35	0.707
Post ME 3Ch strain	-10.59 ± 4.2				
ME 4Ch strain					
Pre ME 4Ch strain	-9.91 ± 2.83	0.95	0.71	2.62	0.247
Post ME 4Ch strain	-10.86 ± 3.51				
Propofol (n=21)					
GLS					
Pre-CPB GLS	-12.52 ± 2.36	0.62	0.16	1.40	0.114
Post-CPB GLS	-11.9 ± 2.49				
ME 2Ch strain					
Pre ME 2 Ch strain	-14.48 ± 4.64	2.38	0.77	3.99	0.006*
Post ME 2 Ch strain	-12.1 ± 3.91				
ME 3Ch strain (LAX)					
Pre ME 3Ch strain	-11.1 ± 2.98	0.38	0.80	1.56	0.509
Post ME 3Ch strain	-11.48 ± 3.11				
ME 4Ch strain					
Pre ME 4Ch strain	-12.43 ± 2.99	0.19	1.83	2.21	0.846
Post ME 4Ch strain	-12.62 ± 3.87				

Table 20 comparing GLS and related parameters between pre-CPB and post-CPB periods within study groups showed a statistically significant decrease in ME 2Ch strain in propofol group. There was no difference that was statistically significant in any other parameters compared.

Table 21: Comparison of mean of change in GLS, GCS and regional parameters between study groups(n=43)

Parameter	Study Groups (Mean± SD)		*p-value*
	Sevoflurane (N=22)	Propofol (N=21)	
Change in GLS	-0.27 ± 2.43	0.62 ± 1.72	0.174
Change in ME 2Ch strain	0.86 ± 4.78	2.38 ± 3.53	0.245
Change in ME 3Ch strain	-0.36 ± 4.48	-0.38 ± 2.6	0.988
Change in ME 4Ch strain	-0.95 ± 3.76	-0.19 ± 4.43	0.545
Change in GCS	-3.23 ± 4.3	-1.33 ± 3.57	0.125
Change in TG basal Strain	-2.86 ± 3.24	-1.24 ± 2.49	0.073
Change in TG mid Strain	-3.59 ± 5.39	-0.05 ± 6.22	0.052
Change in TG apical Strain	-2.64 ± 6.99	-4.86 ± 7.53	0.322

Table 22 showing no significant change between sevoflurane and propofol group in GLS, GCS and regional strain values post AVR.

Table 22: Correlation between pre-CPB GLS and VIS on arrival in ICU in the study population (n = 43)

Parameter	Pearson's Correlation (r)	*p-value*
VIS on arrival in ICU vs pre-CPB GLS	-0.290	0.059
VIS on arrival in ICU vs pre-CPB GCS	0.277	0.072

Table 22 depicts weak negative correlation between VIS on arrival in ICU and pre-CPB GLS. There is also a weak positive correlation between VIS on arrival in ICU and pre-CPB GCS.

Figure 9 : Scatter bar chart of comparison of VIS Vs Pre-CPB GLS (n=43)

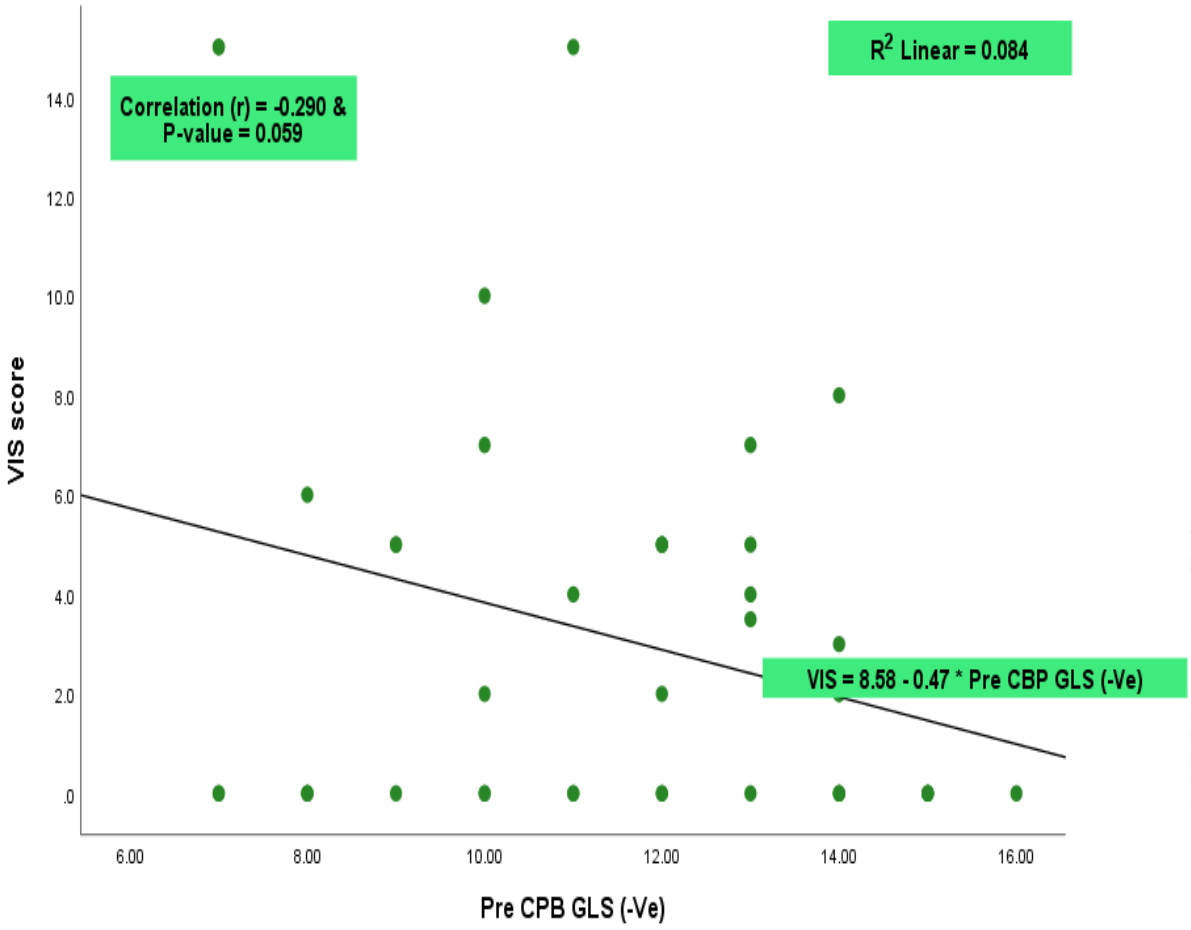


Figure 9 showing weak negative correlation between pre-CPB GLS and VIS on arrival in ICU that is not statistically significant.

Pearson correlation co-efficient [r]:- Positive value denotes direct correlation whereas negative value signifies inverse correlation. (0 to 0.35- poor/weak correlation, 0.36 to 0.55- good correlation, > 0.55 significant correlation.

Figure 10: Scatter bar chart of comparison of VIS Vs Pre-CPB GCS (n=43)

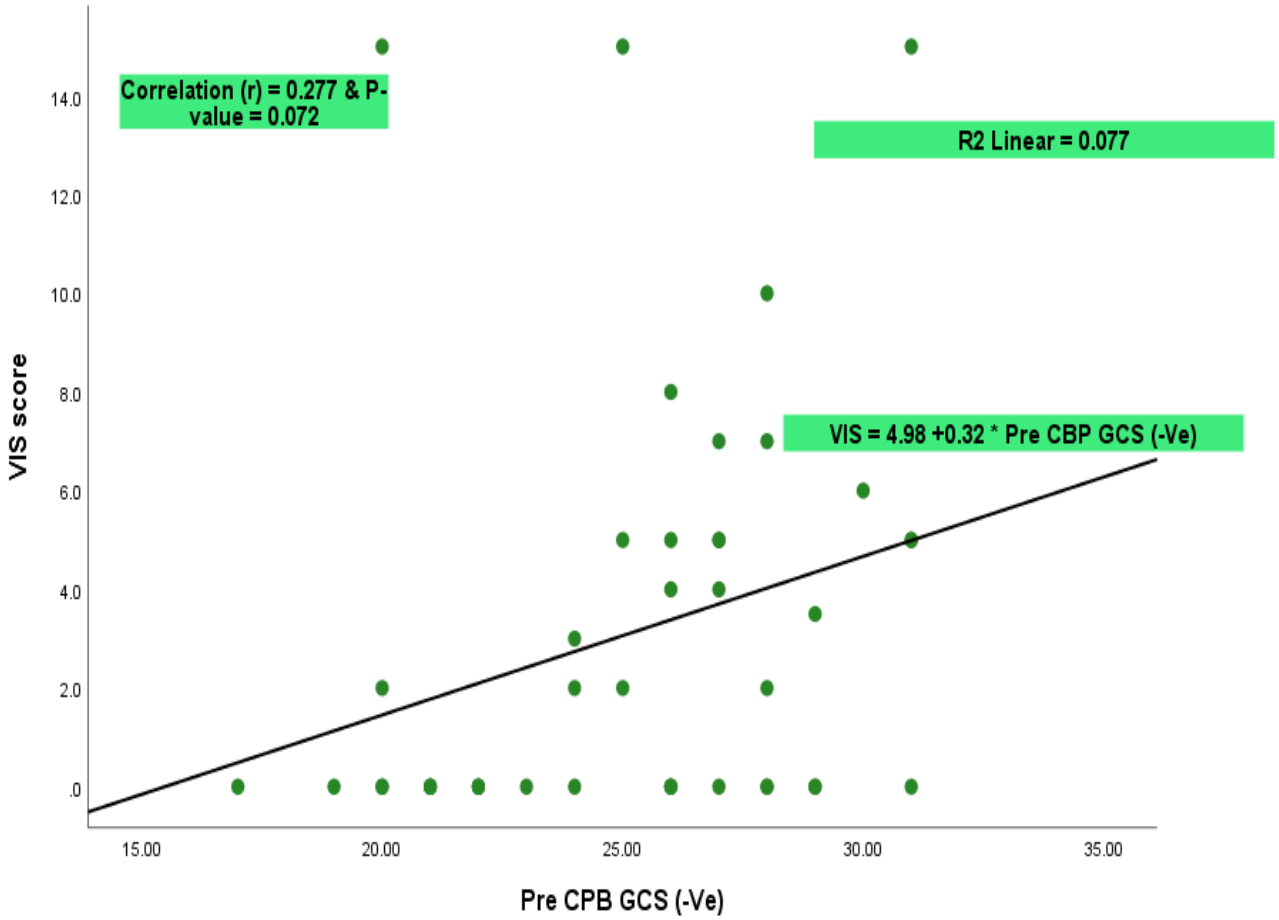


Figure 10 showing weak positive correlation between pre-CPB GCS and VIS on arrival in ICU that is not statistically significant.

Pearson correlation co-efficient [r]:- Positive value denotes direct correlation whereas negative value signifies inverse correlation. (0 to 0.35- poor/weak correlation, 0.36 to 0.55- good correlation, > 0.55 significant correlation.

Table 23: Comparison of mean of pre-CPB GLS, pre-CPB GLS & VIS between LV mass index (gm/m^2) (n=43)

Parameter	LV MASS INDEX (gm/m^2) (Mean± SD)		P value
	LVH (n=35)	No LVH (n=8)	
Pre CPB GLS	-11.31 ± 2.64	-12 ± 2.56	0.510
Pre CPB GCS	-25.2 ± 3.87	-25.63 ± 2.83	0.772
VIS on ICU arrival	3.3 ± 4.49	2.5 ± 3.3	0.638

Table 22 depicting comparison of mean of pre-CPB GLS, GCS and VIS on ICU arrival in patients with and without LVH. There were no statistically significant differences in above parameters between patients having or not having LVH. LVH was defined as LV mass index > 115 gm/m^2 in men and more than 95 gm/m^2 in women.

Abbreviations:- LVH: Left ventricular hypertrophy.

Table 24: Intra-class correlation coefficient (measure of reliability) assessment of GLS, GCS and regional strain values by baseline values and intra-observer values (n=10)

	Intra class Correlation	95% CI		*p-value*
		Lower bound	Upper bound	
Pre-CPB GLS	0.930	0.746	0.982	<0.001*
Pre ME 2Ch strain	0.909	0.677	0.977	<0.001*
Pre ME 3Ch strain	0.768	0.308	0.937	0.003*
Pre ME 4Ch strain	0.690	0.151	0.913	0.009*
Pre-CPB GCS	0.973	0.894	0.993	<0.001*
Pre TG basal	0.940	0.777	0.985	<0.001*
Pre TG mid	0.811	0.409	0.949	0.001*
Pre TG apical	0.904	0.662	0.975	<0.001*
Post-CPB GLS	0.975	0.904	0.994	<0.001*
Post ME 2Ch strain	0.928	0.739	0.982	<0.001*
Post ME 3Ch strain	0.889	0.617	0.971	<0.001*
Post ME 4Ch strain	0.817	0.424	0.951	0.001*
Post-CPB GCS	0.959	0.846	0.990	<0.001*
Post TG basal	0.759	0.288	0.934	0.003*
Post TG mid	0.938	0.773	0.984	<0.001*
Post TG apical	0.726	0.221	0.924	0.006*

Table 24 showing intra class correlation coefficient between baseline strain values and values measured by same observer after one month (intraobserver variability) that revealed statistically significant good correlation between pre-CPB GLS, pre-CPB GCS, post-CPB GLS and post-CPB GCS values.

Intra-class correlation coefficient between 0.7 to 0.9 is good correlation.

* indicates statistically significant values.

Table 25: Inter-class correlation coefficient (measure of reliability) assessment of GLS, GCS and regional strain values by baseline strain values and inter-observer values (n=10)

	Inter class Correlation	95% CI		*p-value*
		Lower bound	Upper bound	
Pre-CPB GLS	0.948	0.805	0.987	<0.001*
Pre ME 2Ch strain	0.420	0.215	0.689	0.043*
Pre ME 3Ch strain	0.675	0.123	0.908	0.011*
Pre ME 4Ch strain	0.596	-0.010	0.882	0.026*
Pre-CPB GCS	0.972	0.893	0.993	<0.001*
Pre TG basal	0.964	0.863	0.991	<0.001*
Pre TG mid	0.876	0.580	0.968	<0.001*
Pre TG apical	0.861	0.537	0.964	<0.001*
Post-CPB GLS	0.993	0.973	0.998	<0.001*
Post ME 2Ch strain	0.981	0.925	0.995	<0.001*
Post ME 3Ch strain	0.978	0.916	0.995	<0.001*
Post ME 4Ch strain	0.974	0.898	0.993	<0.001*
Post-CPB GCS	0.981	0.928	0.995	<0.001*
Post TG basal	0.777	0.329	0.940	0.002*
Post TG mid	0.898	0.645	0.974	<0.001*
Post TG apical	0.922	0.720	0.980	<0.001*

Table 24 showing interclass correlation coefficient between baseline strain values and values measured by different observer (interobserver variability) that revealed statistically significant good correlation between pre-CPB GLS, pre-CPB GCS, post-CPB GLS and post-CPB GCS values.

Inter-class correlation coefficient between 0.7 to 0.9 is good correlation.

* indicates statistically significant values.

DISCUSSION

DISCUSSION

In our prospective randomized study, we compared sevoflurane with propofol in patients of severe AS with good LVEF undergoing AVR, based on intraoperative changes in GLS and GCS before and after AVR. Patients included in study were administered sevoflurane or propofol anesthesia as per group allotted, which was titrated to BIS value in range of 40 to 50. Patients with severe AS and LVEF more than 50% in sinus rhythm for elective AVR were included in our study. Other factors that could possibly affect GLS and GCS calculation, like presence of any other valvular lesion that was more than mild, patients on preoperative inotropes, patients with CAD with lumen stenosis more than 50% in at least one of major epicardial coronaries and patients with poor endocardial differentiation on echocardiography were excluded from our study. Preload, contractility and afterload related factors that may affect GLS and GCS values were kept within 20 % of baseline throughout the study period. Most of these variables were statistically found to be not different between sevoflurane and propofol group. GLS and GCS with corresponding regional strain values together with other 2D echocardiographic and doppler derived parameters were compared between groups and within the group in pre-CPB and post-CPB period. Statistical evaluation was also done to analyze changes in GLS and GCS values post AVR. Preoperative risk factors that may have a bearing on preoperative GLS and GCS values were also interrogated in our study(46,47)

Factors affecting LV systolic function

There are many factors that can affect the LV systolic function during perioperative period in severe AS patients with good LVEF undergoing AVR. Various baseline characteristics of patient such as age, gender, preoperative risk factors, perioperative echocardiographic parameters and aortic cross clamp time are known to influence perioperative LV systolic function in severe AS (46,47). Our study revealed most of these factors to be similar between sevoflurane and propofol group. In addition, all patients recruited for the study were in sinus

rhythm, that avoided the effect of AF on LV systolic function in severe AS patients (48). All other drugs used for induction and maintenance of anesthesia like opioids and benzodiazepines, which could affect perioperative hemodynamics in these patients were used in equivalent doses in both groups as per study protocol to avoid its confounding effects. Thus, any perioperative changes in LV systolic function which reflected as changes in GLS and/or GCS values between groups, were solely attributed to the choice of anesthetic regimen.

Limitations of conventional echocardiographic parameters for evaluation of LV systolic function

Conventional echocardiographic techniques that have been used by many studies comparing sevoflurane with propofol on LV systolic function have many limitations. EF, which is the commonly used echocardiographic parameter to assess LV systolic function is load dependent and needs accurate tracing of endocardial border (12). EF calculation is also based on certain geometrical assumptions that may not be universal in severe AS patients (12). Ejection fraction is also a late marker of systolic dysfunction in severe AS patients which can cause a delay in timely valve replacement. When compared with EF, GLS and GCS values detects early systolic dysfunction in severe AS (17) patients and is of prognostic value (2,32).

Techniques for strain quantification in perioperative period

There are several techniques available for strain quantification like TDI, 2D STE and MRI based techniques. The major issue with use of TDI for strain calculation in perioperative period is its angle dependency that could cause errors in analyzing of mid and apical segments (21). MRI based strain calculation, although considered a gold standard for strain quantification, is not practically feasible in intraoperative period. Hence, we utilized 2D STE quantification which is angle-independent, less influenced by translation and tethering artifacts (21). It can provide GLS and GCS values together with real-time regional strain parameters in operation room. 2D STE quantification has been found to correlate with MRI based EF which is regarded as the gold standard for LV

systolic function evaluation (31). Our study found that GLS and GCS with regional strain parameters had good sensitivity in detecting early systolic dysfunction in severe AS patients presenting for AVR with an apparently good LVEF.

The main observations from our study are as below

1. There were no statistically significant differences in demography, preoperative risk factors and preoperative TTE-derived echocardiographic and doppler parameters between sevoflurane and propofol group.

2. There were no statistically significant differences in pre-CPB hemodynamic parameters and TEE-derived echocardiographic and doppler parameters between sevoflurane and propofol group.

3. Pre-CPB GLS, ME 4Ch and ME 2Ch regional strain values were found to be more in propofol group when compared to sevoflurane group. Pre-CPB regional longitudinal strain values were observed to be more in apical segments when compared to basal segments in both groups.

4. Sevoflurane group had higher values of TG basal strain when compared to propofol group in pre-CPB period. There was no difference in pre-CPB GCS and regional circumferential strain between groups. There were inconsistent differences in regional strain values at basal and apical regions, as against that was seen with regional longitudinal strain.

5. Patients who were preoperatively on calcium channel blockers were found to be having a statistically significant higher pre-CPB GCS values when compared to those not receiving them. Similarly, patients on ARB/ACEI were found to be having lower pre-CPB GLS values vis a vis patient not treated with them.

Preoperative hypertension, diabetes mellitus and beta blocker usage were found to generate any significant impact on global longitudinal and circumferential myocardial function.

6. Aortic cross clamp time, prosthetic valve size and type were alike among the study groups. CPB time was higher in sevoflurane group than propofol group. Both study groups were comparable in terms of the VIS score during CPB-weaning.

7. There was no difference in post-CPB hemodynamic, 2D TEE and doppler parameters between the study groups on weaning from CPB.

8. Post-CPB GLS, GCS and corresponding regional strain values were found to be similar between groups.

9. VIS on arrival in ICU was found to be higher in sevoflurane group. However, no differences were observed in duration of ventilation, ICU stay and postoperative TTE- parameters.

10. There was no difference in GLS values in pre-CPB and post-CPB period across study groups. GCS and corresponding regional strain values were significantly lower in the post-CPB period when compared to pre-CPB across study groups. This reduction in GCS and corresponding segmental values were markedly significant in sevoflurane group. There was no difference between sevoflurane and propofol groups in mean of change in GLS, GCS and regional strain parameters when post-CPB values were compared with pre-CPB values.

11. EF in preoperative and postoperative period was found to be similar between groups.

12. A weak negative correlation was found among pre-CPB GLS and VIS on arrival in ICU. Similarly, a weak

positive correlation was observed between pre-CPB GCS and VIS during same period. Pre-CPB GLS, GCS and VIS on arrival in ICU were found to be similar in patients with or without LVH.

Preoperative baseline GLS and GCS in severe AS patients with good LVEF

Many studies have been published evaluating baseline GLS and GCS in severe AS patients with good LVEF. We observed low baseline GLS and high baseline GCS values in our study patients. This finding corresponds to study by Carasso et al (3) who studied myocardial mechanics in severe AS patients with good LVEF undergoing AVR. However, Delgado et al (1) in their study reported that both GLS and GCS values were significantly decreased in same subset of patients. Kaler et al (49) and Diana et al (50) had similar observations.

The reason for this dichotomy observed between studies is possibly the natural progress of strain values in severe AS, as reported by Carasso et al (3) . It was postulated by them that increase in the circumferential strain values compensated for decrease in longitudinal strain values in early phases of severe aortic stenosis. This compensatory response in circumferential strain was lost as the disease progressed in severity, with resultant decline in circumferential strain values.

Effect of preoperative risk factors on pre-CPB GLS and GCS

There are several risk factors that are known to influence the progression of LV dysfunction in severe AS patients. Published literature (51,52) has identified many of these risk factors like diabetes, hypertension etc. We contemplated evaluating the effect of these known preoperative risk factors on baseline GLS and GCS, as strain quantification is known to detect subtle systolic dysfunction earlier than routine echocardiographic parameters (17).

We ascertained the association between preoperative risk factors such as hypertension, diabetes mellitus, ACEI/ARB, CCB and beta blocker therapy on pre-CPB GLS and GCS values in our study groups. We discovered

that prior CCB treatment is associated with higher GCS values. Coincidentally, preoperative ACEI/ARB treatment was found to be associated with lower GLS scores compared to those who did not receive that treatment.

It is an established fact that hypertension in presence of severe AS induces a 'twin pressure load' on the left ventricle, adversely affecting LV function and increasing mortality (53). We presume that treatment of hypertension with CCB decreases the LV afterload which correspondingly curtail the stress on circumferential LV fibres and augmenting pre-CPB GCS values.

On the other hand, as ACEI and ARB are administered to severe AS patients who have preexisting symptomatic heart failure, lesser GLS values are expected (54). We opine that the association of ACEI/ARB usage with low pre-CPB GLS values was attributable to the preexistent heart failure with preserved ejection fraction rather than the effect of ACEI/ARB on longitudinal fibres.

To the best of our knowledge, we did not find any literature evaluating effects of ACEI/ARB and CCB on GLS and GCS respectively in patients with AS.

Intraoperative changes in GCS post-AVR in severe AS patients with preserved LVEF

One of the primary objectives of our study was to observe intraoperative changes in GLS and GCS in severe AS patients with good LVEF undergoing AVR. Most of the published literature in this area is focused on post operative follow-up ranging from 7 days to 7 months after the surgery. Our study objective was to analyze the changes in GLS and GCS in immediate postoperative period. However, the literature search revealed scanty evidence on perioperative changes in GLS and GCS in patients with severe AS.

The study done by Carasso et al (3) on 32 patients to evaluate myocardial mechanics pre-AVR and post-AVR in severe AS patients with good LVEF revealed lower preoperative GLS values with compensatory higher values for GCS. The authors found that the GLS increases and the GCS decreases a week after replacement of the diseased

valve. However, Kaler et al (49) in 70 patients with severe AS noted that both GLS and GCS values were reduced in the preoperative period. The GCS values continued to abate 1 week after the AVR. These changes in the strain quantification however were short lasted as higher GLS and the GCS values were found at 3 months after AVR suggesting a recovery of the longitudinal and circumferential myocardial function. Consistent with the previous study, Rost et al (55) reported that GLS and GCS values did not change within one week after AVR, albeit these strain values increased significantly within three months. In another study (1) the low GLS and GCS values in the preoperative period significantly improved on follow-up evaluation at 17 months after AVR. In a CMRI derived strain study (50) on patients with severe AS with preserved LVEF, the baseline low GLS and GCS were found to deteriorate further 6 months after the AVR. However, the radial strain did not alter significantly.

Most of our patients had low GLS values and high GCS values in preoperative period that concurs with findings by Carasso et al (3) and Diana et al (50). The cause of decrease in the GCS in our study population is possibly the abrupt decrease in afterload secondary to relief of aortic stenosis and generalized vasodilatory state in the post-bypass period.

Intraoperative changes in GLS post-AVR in severe AS patients with preserved LVEF

In contrast to findings by Carasso et al (3), GLS values were not found to increase in immediate postoperative period in our study population. Similar observations were also reported in other studies(49,55). There are multiple factors that could have led to this mismatch. Post-AVR strain analysis was done in our patients at the time of sternotomy and hence the overt effects of CPB on myocardial function are expected. Labus et al(33) in their study on 2D STE in on-pump CABG patients had observed that postoperative GLS values were less when compared to preoperative counterparts, which they attribute to the adverse myocardial effects of CPB. Worsening of GLS in post-CPB period in our study subjects could be because of adverse myocardial effects of CPB, as congruent with the other studies(55–58). Further, CPB may cause myocardial dysfunction lasting for about a month after cardiac surgery which may explain our observations (57).

We also found that despite changes in GCS values post- AVR, there was no significant change in EF between preoperative and postoperative periods. This is in concurrence with studies done by Delgado et al (1), Rost et al (55) and Diana et al (50) that further strengthens utility of GLS and GCS for evaluating subtle changes in LV systolic function in perioperative period.

To the best of our knowledge, scanty literature is available evaluating changes in GLS and GCS in immediate post AVR period in severe AS patients as ascertained by us. The replacement of diseased aortic valve results in lowering the LV afterload which manifests as decrease in the GCS. Extrapolating the GCS-reducing consequences of AVR to the evaluation of prosthetic valve area, we propose that GCS lowering effects would be expected only in the absence of patient prosthesis mismatch. However, as we did not encounter any patient with patient prosthetic mismatch, we could not verify our proposition.

Further, post-CPB GLS did not differ with pre-CPB values which suggest that a minimum period is necessary for the myocardial systolic function to recover and the reverse remodeling to take place after on-pump cardiac surgery. We opine that the GLS values may not alter in the immediate post-surgical period; hence, many weeks must elapse before the GLS changes to occur after valve replacement in severe AS patients with good LVEF. The same may also hold true in postoperative GLS quantification after other on-pump cardiac surgeries.

Effect of volatile agents and propofol on commonly measured echocardiographic parameters

Volatile agents and Propofol when used in higher concentration, are known to cause hemodynamic changes.
Both drugs are known to cause arteriolar dilation which is more pronounced on induction with propofol. There are many studies that oscillates towards the beneficial effects of volatile anesthesia regimen when compared with

propofol regimen in cardiac surgery(34,35,40,41); whereas, many other studies demonstrate equivocal results(36–39,42). As these agents were expected to cause variable changes in intraoperative myocardial function, we expected it to get reflected in transesophageal echocardiographic parameters. In our study no differences were noted with respect to commonly measured echocardiographic parameters between sevoflurane and propofol group. It agrees with study by Lindholm et al(59) who evaluated transthoracic echocardiographic parameters between volatile and TIVA regimen in 164 vascular surgery patients. Chennakeshavullu(4) in his study comparing effect of strain quantification between sevoflurane and propofol, also reported similar findings.

The reason for similarity in conventional echocardiographic parameters between groups in our study is possibly due to the titration of anesthetic depth in both groups to a standard BIS value of 40 to 50. Similar VIS score while weaning from CPB in both the groups could be another reason why we did not encounter diverse echocardiographic parameters. In addition, we ensured maintaining preload, contractility and afterload within +/- 20% of baseline for all our cases throughout study period which minimized the differences arising from hemodynamic parameters.

Effect of sevoflurane and propofol on changes in GLS and GCS post-AVR

Global and regional strain quantification was used to compare effects of sevoflurane and propofol anesthesia on LV systolic function in our study population. Analysis was performed comparing GLS, GCS and segmental strain values between both groups in pre-CPB and post-CPB period.

In pre-CPB period, propofol group was found to be having significantly higher value of GLS, mainly in regional ME 2Ch and ME 4Ch regions. There were no major differences found between GCS values between both groups in pre-CPB period. In post-CPB period there were no differences between GLS and GCS values between sevoflurane and propofol group.

There is paucity of literature comparing sevoflurane and propofol anesthesia based on strain quantification. Study done by Chennakeshavullu et al(4) compared sevoflurane with propofol anesthesia in on-pump CABG patients and reported no difference in GLS and GCS between groups. Another study by Coleman et al(5), who compared GLS values before and after propofol induction in endoscopic procedures reported that propofol induction does not significantly alter the GLS values from baseline. Our study results were similar to Chennakeshavullu et al(4) as we did not find any difference in change of mean GCS and GLS values between sevoflurane and propofol post-AVR.

We also found that longitudinal segmental strain values were more in apical regions when compared to basal regions which is in concurrence with other studies (60) (23)

Pre-CPB GLS and GCS and its effect on VIS on arrival in ICU

We wanted to analyze if there was any relation between pre-CPB GLS, pre-CPB GCS and VIS on arrival in ICU. The mean VIS score on arrival in ICU in sevoflurane group was 4.6 +/- 5.2 when compared to 1.6 +/- 2.22 in propofol group that was found to be statistically significant. It may be possible that the vasodilatory effect of sevoflurane persists for a longer duration.

We found a weak negative correlation between pre-CPB GLS and VIS on arrival in ICU. Similarly, a weak positive correlation was found between pre-CPB GCS and VIS on arrival in ICU. Zhang et al (32) reported that lower preoperative GLS was associated with prolonged hospital stay and increased inotrope requirement in cardiac surgery.

We expected that low GLS and high GCS values in our patients would demonstrate a strong positive correlation with VIS on arrival in ICU because of subtle worsening in LV systolic function.

We are not certain about the changes taking place in radial strain quantification after AVR. Radial strain values in normal range (61) was reported in pre-AVR period by Rost et al(55) which they reported to increase significantly 6 month after AVR. Hence, the weak correlation between pre-CPB GLS and GCS with postoperative VIS whether could possibly be attributed to the changes in the radial strain remains an area to be explored in our opinion.

Limitations of our study

1. Radial strain calculations were not available in the software package that we utilized for strain quantification. Hence, we couldn't account for changes in radial strain that could have helped in making a clearer analysis of our findings.

2. We could not achieve our target sample size because of covid related OT closures followed by decreased number of surgeries that adversely affected the power of our analysis. We may consider in future to conduct a reanalysis of results once the target sample size is achieved.

3. We excluded patients who had atrial fibrillation and those who required cardiac pacing in the post-CPB period as our software was unable to perform strain analysis in these subsets of patients.

4. 2/3rd of our patients were on minimal dose of inotropes and/or vasopressors in post-CPB period which were continued on arrival in ICU. Inotrope and vasopressor infusions could have influenced the strain values.

5. Normal values for GLS and GCS varies with different software packages provided by various vendors. Hence, our results may not be applicable in toto with packages from other vendors.

SUMMARY
AND
CONCLUSION

SUMMARY AND CONCLUSION

In our study we compared GLS values and GCS values in the preoperative and the postoperative period between sevoflurane regimen and propofol regimen. Our conclusions were as follows

Primary objectives

1. In the pre-CPB period, when GLS were compared between sevoflurane and propofol group, the GLS was found better (more negative) in propofol group than sevoflurane group. No differences were observed in GCS between propofol group and sevoflurane group in pre-CPB period.

2. In the post-CPB period, when GCS were compared between sevoflurane and propofol group, no differences were found between the groups in GLS as well as GCS.

3. When the sample study subjects were put together (n=43), and pre-CPB GLS were compared with post-CPB GLS, we found no differences.

4. When the sample study subjects were put together (n=43), and pre-CPB GCS were compared with post-CPB GCS, we found that post-CPB GCS was less than pre-CPB GCS.

5. When pre-CPB GLS were compared with post-CPB GLS in sevoflurane group, we found no difference. Similarly, when pre-CPB GLS were compared with post-CPB GLS in propofol group, there were no differences.

6. When pre-CPB GCS were compared with post-CPB GCS in sevoflurane group, we observed that the GCS value reduced (less negative) in sevoflurane group. However, when pre-CPB GCS were compared with post-CPB GCS in propofol group, no differences were observed

7. When we compared combined pre-CPB and post-CPB values for changes in the GLS in sevoflurane group with those of propofol group, we found no statistically significant difference.

8. When we compared combined pre-CPB and post-CPB values for changes in the GCS in sevoflurane group with those of propofol group, we found no statistically significant difference.

Secondary objectives

1. The mean reference values for GLS and GCS were considered as published in JASE (reference 61), which were 19.7% and 23.3%, respectively. We observed in our study that GLS values combined for both sevoflurane and propofol group in the pre-CPB period in patients with severe AS with good LVEF were less, whereas the GCS values were increased.

2. Patients who were on CCB were found to be having significantly increased GCS values pre-CPB when compared to those not on them. Similarly, patients on ACEI/ARB were found to be having significantly less GLS score preoperatively when compared to those not using them.

ANNEXURES

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STUDY PROFORMA

NAME:

AGE: WEIGHT:

GENDER: HEIGHT:

HOSPITAL NUMBER: BSA:

DIAGNOSIS: DATE:

ANAESTHESIA TECHNIQUE: SEVOFLURANE GROUP/ PROPOFOL GROUP

PRE-OPERATIVE DATA

HISTORY OF HYPERTENSION	
HISTORY OF DIABETES MELLITUS	
CALCIUM CHANNEL BLOCKER USE	
BETA BLOCKER USE	
ACE IHIBITOR / ARB USE	
SEPTAL WALL THICKNESS SYS/DIAS	
INFEROLATERAL WALL THICKNESS SYS/DIAS	
AS MEAN GRADIENT	
AS PEAK VELOCITY	
EJECTION FRACTION	
REMARKS	

PRE-CPB DATA

HEART RATE		
BP SYSTOLIC/DIASTOLIC (MEAN)		
PULSE PRESSURE		
CVP LVEDA		
LVEF		
LVOT CSA		
LVOT VTI		
LVOT SV		

LVOT SV INDEX		
LVOT CARDIAC OUTPUT		
SVR (MAP – CVP)/CO x 80		
SYSTEMIC ARTERIAL COMPLIANCE (SVI/PULSE PRESSURE)		
VALVULOARETERIAL IMPEDANCE (SYSTOLIC BP + MEAN GRADIENT)/SVI		
SEPTAL THICKNESS		
POSTERIOR WALL THICKNESS		
LVIDD		
RELATIVE WALL THICKNESS (2 x POSTERIOR WALL THICKNESS)/LVIDD		
LV LONG AXIS LENGTH		
LV MASS		
LV MASS INDEX		
LA SIZE		
E/E'		
GLOBAL LONGITUDINAL STRAIN		
TWO CHAMBER VIEW	BASAL ANTERIOR	
	MID ANTERIOR	
	APICAL ANTERIOR	
	BASAL INFERIOR	
	MID INFERIOR	
	APICAL INFERIOR	
	APEX	
	TWO CHAMBER STRAIN	
THREE CHAMBER VIEW	BASAL ANTEROSEPTAL	
	MID ANTEROSEPTAL	
	APICAL ANTEROSEPTAL	
	BASAL INFEROLATERAL	
	MID INFEROLATERAL	
	APICAL INFEROLATERAL	
	APEX	
	THREE CHAMBER STRAIN	

FOUR CHAMBER VIEW	BASAL ANTEROLATERAL	
	MID ANTEROLATERAL	
	APICAL ANTEROLATERAL	
	BASAL INFEROSEPTAL	
	MID INFEROSEPTAL	
	APICAL INFEROSEPTAL	
	APEX	
	FOUR CHAMBER STRAIN	
GLOBAL CIRCUMFERENTIAL STRAIN		
TRANSGASTRIC BASAL VIEW	INFERIOR	
	INFEROSEPTAL	
	ANTERIOSEPTAL	
	ANTERIOR	
	ANTEROLATERAL	
	INFEROLATERAL	
	TRANSGASTRIC BASAL STRAIN	
TRANSGASTRIC MID VIEW	INFERIOR	
	INFEROSEPTAL	
	ANTERIOSEPTAL	
	ANTERIOR	
	ANTEROLATERAL	
	INFEROLATERAL	
	TRANSGASTRIC MID STRAIN	
TRANSGASTRIC APICAL VIEW	INFERIOR	
	SEPTAL	
	ANTERIOR	
	LATERAL	
	TRANSGASTRIC APICAL STRAIN	
REMARKS		

CPB DETAILS

AORTIC CROSS CLAMP TIME	
CPB TIME	
REMARKS	

SURGICAL DETAILS

TYPE OF VALVE	
SIZE OF VALVE	
REMARKS	

POST CPB

INOTROPES	
VASOCONSTRICTORS	
VASODILATORS	
POST CPB HEMOGLOBIN	
CVP LVEDA	
HEART RATE	
PACING	
REMARKS	

POST CPB DATA (AFTER STERNAL CLOSURE)

AORTIC VALVE PROSTHESIS PEAK VELOCITY		
AV MEAN GRADIENT		
ACCELERATION TIME		
EFFECTIVE ORIFICE AREA INDEX		
DVI		
ANY PARAVALVULAR LEAK		
LVOT CARDIAC OUTPUT		
TAPSE		
SVR		
GLOBAL LONGITUDINAL STRAIN		
TWO CHAMBER VIEW	BASAL ANTERIOR	
	MID ANTERIOR	
	APICAL ANTERIOR	
	BASAL INFERIOR	
	MID INFERIOR	
	APICAL INFERIOR	
	APEX	
	TWO CHAMBER STRAIN	

THREE CHAMBER VIEW	BASAL ANTEROSEPTAL	
	MID ANTEROSEPTAL	
	APICAL ANTEROSEPTAL	
	BASAL INFEROLATERAL	
	MID INFEROLATERAL	
	APICAL INFEROLATERAL	
	APEX	
	THREE CHAMBER STRAIN	
FOUR CHAMBER VIEW	BASAL ANTEROLATERAL	
	MID ANTEROLATERAL	
	APICAL ANTEROLATERAL	
	BASAL INFEROSEPTAL	
	MID INFEROSEPTAL	
	APICAL INFEROSEPTAL	
	APEX	
	FOUR CHAMBER STRAIN	
GLOBAL CIRCUMFERENTIAL STRAIN		
TRANSGASTRIC BASAL VIEW	INFERIOR	
	INFEROSEPTAL	
	ANTERIOSEPTAL	
	ANTERIOR	
	ANTEROLATERAL	
	INFEROLATERAL	
	TRANSGASTRIC BASAL STRAIN	
TRANSGASTRIC MID VIEW	INFERIOR	
	INFEROSEPTAL	
	ANTERIOSEPTAL	
	ANTERIOR	
	ANTEROLATERAL	
	INFEROLATERAL	
	TRANSGASTRIC MID STRAIN	
TRANSGASTRIC APICAL VIEW	INFERIOR	
	SEPTAL	
	ANTERIOR	
	LATERAL	
	TRANSGASTRIC APICAL STRAIN	

REMARKS	

POST OPERATIVE PARAMETERS

VASOPRESSOR INOTROPE SCORE	
DURATION OF ARTIFICIAL VENTILATION (HOURS)	
ICU STAY (HOURS)	
PEAK VELOCITY	
MEAN GRADIENT	
LV EJECTION FRACTION	
REMARKS	

IEC APPROVAL

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/RR-21)

SCT/IEC/1843/FEBRUARY/2022 25.04.2022

Dr. Markose L Paret
Senior Resident
Department of Anaesthesiology
SCTIMST, Thiruvananthapuram

Dear Dr. Markose L Paret,

The Institutional Ethics Committee held on 19^{in} February, 2022, reviewed and discussed your application to conduct the study titled "**EFFECT OF SEVOFLURANE VERSUS PROPOFOL ON STRAIN QUANTIFICATION IN PATIENTS WITH SEVERE AORTIC STENOSIS UNDERGOING AORTIC VALVE REPLACEMENT** " (IEC/1843).

The following members of the Ethics Committee were present at the meeting held on 19th February, 2022

SL. No.	Member Name	Highest Degree	Gender	Scientific /Non Scientific	Affiliation with Institution(s)
1.	Prof. C.C. Kartha	MBBS,MD	Male	Basic Medical Scientist (Chairman)	No
2.	Dr. Kala Kesavan P	MBBS,MD	Female	Basic Medical Scientist	No
3.	Smt. Sathi Nair	MA (English Literature)	Female	Lay Person	No
4.	Dr. Pradeep S	MBBS, MD	Male	Basic Medical Scientist	No
5.	Dr. P. Manickam	BSMS, MSc (Epid).,PhD	Male	Health Science Expert/ Social Scientist	No
6.	Dr. Christina George	MD Psychiatry	Female	Clinician	No
7.	Adv. N Anand	BAL, L.LB	Male	Legal Expert	No
8.	Adv. Priya Kaimal	LLM, MBL	Female	Legal Expert	No
9.	Dr. Harikrishna Varma PR	Ph.D (Materials Science)	Male	Medical Technology	Yes
10.	Dr. Narayanan Namboodiri. K K	MBBS,MD,DM	Male	Clinician	Yes
11.	Dr. Ashalatha R	MBBS, MD,DM	Female	Clinician	Yes
12.	Dr. Biju Soman	MBBS,MD, DPH, MSc, DLSHTM	Male	Basic Medical Scientist	Yes
13.	Dr. Srinivas G	PhD	Male	Basic Medical Scientist (Member Secretary)	Yes

The following documents were reviewed:

Original submission

1. *Checklist Form*
2. *Project Proposal*
3. *Data Collection Proforma*
4. *IEC Application Form*
5. *Covering letter addressed to the Chairman, IEC, SCTIMST*
6. *Data Collection Proforma*
7. *TAC Approval Letter*
1. *Consent Form in English and Malayalam*
2. *CV of PI and Co-PIs*
3. *Covering letter addressed to the Chairman, IEC, SCTIMST*
4. *Dean's signature form*
5. *Declaration Form*
6. *DSMB Members list*

Revised submission

1. *Covering letter addressed to the Chairman, IEC, SCTIMST dated 12.04.2022*
2. *Checklist Form*
3. *Project Proposal*
4. *Data Collection Proforma*
5. *IEC Application Form*
6. *Covering letter addressed to the Chairman, IEC, SCTIMST dated 20.07.2021*
7. *Data Collection Proforma*
8. *TAC Approval Letter*
9. *Consent Form in English and Malayalam*
10. *CV of PI and Co-PIs*
11. *Covering letter addressed to the Chairman, IEC, SCTIMST dated 20.07.2021*
12. *Dean's signature form*
13. *Declaration Form*
14. *DSMB Members list*

IEC Decision

The IEC approved the conduct of the study in the present form.
The study can be started only after getting full CTRI registration.

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,

Dr. G. Srinivas
Member Secretary, IEC

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY
INSTITUTIONAL ETHICS COMMITTEE (IEC)
SCTIMST
THIRUVANANTHAPURAM

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE (IEC)
SCTIMST, THIRUVANANTHAPURAM

CONSENT FORMS

Consent form

Title of the study:

EFFECT OF SEVOFLURANE VERSUS PROPOFOL ON STRAIN QUANTIFICATION IN PATIENTS WITH SEVERE AORTIC STENOSIS UNDERGOING AORTIC VALVE REPLACEMENT

Study numbers: We request you to participate in a study, wherein we are planning to compare the effects of sevoflurane with propofol anesthesia on echocardiographic measurements using intraoperative echocardiographic strain analysis.

What is Sevoflurane or Propofol anesthesia?

Sevoflurane is an inhalational anesthetic agent used in maintenance of general anesthesia. It will be delivered using anesthesia machine, through endotracheal tube commonly placed during all cardiac surgeries, to ensure adequate anaesthesia.

Propofol is an intravenous anesthetic used commonly for induction and maintenance of general anesthesia. This will be delivered intravenously in appropriate concentration using infusion pump to enure adequate anaesthesia.

What are the risks and side effects of Sevoflurane or Propofol Anaesthesia?

The two most commonly used techniques for providing anaesthesia during cardiac surgery is Sevoflurane based technique and Propofol based technique. There are no specific risks with respect to either Sevoflurane or Propofol based anaesthesia during cardiac surgery. Both are equally effective in maintaining adequate anaethesia during cardiac surgery with a proven safety record. In addition, anaesthesia for cardiac surgery is provided to you by experienced cardiac anaesthesiology doctors in SCTIMST with internationally accepted standards of monitoring and care.

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 is helpful in assessing function of heart and 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 ultrasound 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 are the risks and side effects of Transesophageal echocardiography ?

Transesophageal echocardiography is routinely used in patients undergoing cardiac surgery and the risks involved with the use of TEE are negligible. As per 2013 guidelines for transesophageal echocardiography from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists, the complications that can occur due to transesophageal echocardiography are minor bleeding from mouth cavity (0.01%), major bleeding (0.03% to 0.8%), endotracheal tube malposition (0.03%), dental injury (0.03%), severe throat pain (0.1%) and esophageal perforation (0 to 0.3%).

These are risks associated with the use of transesophageal echocardiography and not specific or selective to the study. Transesophageal echocardiography will be done as a part of monitoring and for therapeutic purpose in our hospital for the proposed cardiac surgical procedure irrespective of your participation in this study.

What is the global longitudinal strain?

Global longitudinal strain is a new technique wherein the echocardiographic images acquired during routine transesophageal echocardiohgraphy will be analyzed offline in computer to quantify about cardiac function objectively.

Why are we doing this study?

The purpose of this study is to echocardiographically assess and compare global longitudinal strain, a sensitive and obejective measurement of cardiac function, before and after aortic valve replacement in patients with severe aortic stenosis undergoing aortic valve replacement surgery under sevoflurane or propofol anesthesia. It will also help in understanding risk factors for reduced global longitudinal strain in patients of severe aortic stenosis.

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 but if you develop any side effects or problems due to the study, these complications will be treated at no additional cost to you.

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. Shrinivas V Gadhinglajkar, Professor, Department of Anesthesiology
Tel: 9446304043

Name of the PI:

Dr. Markose L Paret ,Senior Resident, Department of Anesthesiology
Tel: 8853303722 or email id: paretmarcos@gmail.com

For any clarifications regarding the study's ethics clearance you may contact the Member Secretary of SCTIMST-IEC

Dr Srinivas G, (phone number:2524-689, email id : iec.mem.sec@sctimst.ac.in)

Signature and name of the Principal Investigator:

Dr. Markose L Paret

Ph:8853303722

DECLARATION AND INFORMED CONSENT

I_____aged_____(in years/months), _____ (Father/Mother name), give consent to participate in this study titled: ' **EFFECT OF SEVOFLURANE VERSUS PROPOFOL ON STRAIN QUANTIFICATION IN PATIENTS WITH SEVERE AORTIC STENOSIS UNDERGOING AORTIC VALVE REPLACEMENT'**

Please tick the relevant boxes

☐ I confirm that i have read and understood the information sheet dated for the above study and had the opportunity to ask questions to clarify 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 investigators, institutional ethics committee members and regulatory authorities will not need my permission to look at the health records even if I withdraw myself 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 agree not to restrict the use of any data of results that arise from this study provided such a use is only for scientific purposes

☐ I have been given contact numbers to know more about the study and rights of participants in this study

☐ I voluntarily agree to take part in this study

☐ I have received a copy of this signed consent form

Name:

Signature:

Date:

Name of witness:

Relation to participant:

Signature:

Date:

I attest that the requirements for informed consent for the medical research project described in this form have been satisfied. I have discussed the research project with the participant and explained to him or her in nontechnical terms all of the information contained in this informed consent form, including any risks and adverse reactions that may reasonably be expected to occur. I further certify that I encouraged the participant to ask questions and that all questions asked were answered.

Name and Signature of person obtaining consent
Date:

Principal investigator: Dr Markose L Paret
Ph: 8853303722

Witness:

പഠനശീർഷകം: അയോർട്ടിക് വാൽവ് മാറ്റിവയ്ക്കൽ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന ഗുരുതരമായ അയോർട്ടിക് സ്റ്റെനോസിസ് രോഗികളുടെ സ്ട്രെയിനിന്റെ അളവ് നിർണ്ണയത്തിൽ സിവോഫ്ളൂറൈന്റെയും പ്രൊപ്പൊഫോളിന്റെയും പ്രഭാവം തമ്മിലുള്ള താരതമ്യം.

പഠന നമ്പർ: ശസ്ത്രക്രിയാ സമയത്ത് എക്കോകാർഡിയോഗ്രഫിയുപയോഗിച്ച് സിവോഫ്ളൂറൈൻ, പ്രൊപ്പൊഫോൾ എന്നിവയുപയോഗിച്ചുള്ള മയക്കലിന്റെ എക്കോകാർഡിയോഗ്രഫിക് സ്ട്രെയിനിന്റെ താരതമ്യ വിശകലനം നടത്തുന്ന ഒരു പഠനത്തിൽ പങ്കെടുക്കാൻ താങ്കളെ ഞങ്ങൾ ക്ഷണിക്കുന്നു.

സിവോഫ്ളൂറൈൻഅല്ലെങ്കിൽ പ്രൊപ്പൊഫോൾ ഉപയോഗിച്ചുള്ള മയക്കലെന്നാലെന്താണ്?

പൊതുവായ മയക്കലിനും മയക്കം നിലനിർത്താനും ശ്വസിക്കുന്നതിലൂടെ നൽകുന്ന മരുന്നാണ് സിവോഫ്ളൂറൈൻ. ആയത് എല്ലാ ഹൃദയശസ്ത്രക്രിയകളിലും സ്ഥാപിക്കുന്ന എൻഡോട്രാക്കിയൽ കുഴലിലൂടെ അനസ്തേഷ്യ യന്ത്രം വഴി

പൊതുവായ മയക്കലിനും മയക്കം നിലനിർത്താനും കുത്തിവയ്പ്പിലൂടെ നൽകുന്ന മരുന്നാണ് പ്രൊപ്പൊഫോൾ. അത് കുത്തിവയ്പ്പിലൂടെ ആവശ്യമായ സാന്ദ്രതയിൽ ഇൻഫ്യൂഷൻ പമ്പുപയോഗിച്ച് വേണ്ടും വിധമുള്ള മയക്കമുറപ്പാക്കാൻ നൽകും.

സിവോഫ്ളൂറൈൻ അല്ലെങ്കിൽ പ്രൊപ്പൊഫോളുപയോഗിച്ചുള്ള മയക്കലിന്റെ അപായങ്ങളും പാർശ്വഫലങ്ങളുമെന്തെല്ലാം?

ഹൃദയശസ്ത്രക്രിയാവേളയിൽ മയക്കം നൽകുന്നതിന് വളരെ സാധാരണയായി ഉപയോഗിക്കുന്നവയാണ് സിവോഫ്ളൂറൈൻ അടിസ്ഥനമാക്കിയോ പ്രൊപ്പൊഫോൾ അടിസ്ഥാനമാക്കിയോ ഉള്ള സങ്കേതങ്ങൾ. പ്രത്യേകമായ അപായങ്ങളൊന്നും സിവോഫ്ളൂറൈനോ പ്രൊപ്പൊഫോളോ അടിസ്ഥാനമാക്കിയുള്ള മയക്കലിന് ഇല്ല. ഹൃദയശസ്ത്രക്രിയയിൽ അവശ്യമായ മയക്കം നിലനിർത്തുന്നതിന് രണ്ടും തുല്യമായ പ്രഭാവമുള്ളതും തെളിയിക്കപ്പെട്ട സുരക്ഷിതത്വമുള്ളവയുമാണ്. അതോടെപ്പം SCTIMST യിലെ അനുഭവജ്ഞാനമുള്ള ഡോക്ടർമാരാണ് നിരീക്ഷണത്തിന്റെയും പരിചരണത്തിന്റെയും അന്തർദ്ദേശീയമായി അംഗീകരിക്കപ്പെട്ട നിലവാരത്തോടെ താങ്കളുടെ ഹൃദയശസ്ത്രക്രിയയ്ക്കുള്ള മയക്കൽ നൽകുന്നത്.

ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫി എന്നാലെന്ത്?

ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫി അല്ലെങ്കിൽ റ്റിഇഇ ശബ്ദതരംഗങ്ങളുപയോഗിച്ച് ഹൃദയത്തിന്റെയും രക്തധമനികളുടെയും ഉന്നത ഗുണനിലവാരമുള്ള ചലിക്കുന്ന ചിത്രങ്ങൾ നിർമ്മിക്കുന്ന പരിശോധനയാണ്. ഹൃദയത്തിന്റെ പ്രശ്നബാധിതമായ മേഖലകൾ കൃത്യമായി കണ്ടെത്താനും ഹൃദയത്തിന്റെയും ഹൃദയവാൽവുകളുടെയും ശസ്ത്രക്രിയയ്ക്കുമുമ്പും ശേഷവുമുള്ള പ്രവർത്തനം വിലയിരുത്താനും ഇത് സഹായകരമാണ്. സാധാരണയായി എക്കോകാർഡിയോഗ്രഫി ചെയ്യുന്നത് നെഞ്ചുവഴിയാണ് (ട്രാൻസ് തൊറാസിക് എക്കോകാർഡിയോഗ്രഫി). എന്നാൽ, ശസ്ത്രക്രിയാവിദഗ്ദ്ധർ ആ മേഖലയിൽ കൂടിയാണ് ശസ്ത്രക്രിയനടത്തുന്നത് എന്നതിനാൽ ആ സമയത്ത് മയക്കത്തിന് വിധേയമാക്കി, ട്രാൻതൊറാസിക് എക്കോകാർഡിയോഗ്രഫി നടത്താനാവില്ല. ആകയാൽ എക്കോകാർഡിയോഗ്രഫി നിരീക്ഷണ ഉപകരണം താങ്കളുടെ ഈസോഫാഗസ് (അന്നനാളത്തിലൂടെ) കടത്തും. റ്റിഇഇയിൽ അറ്റത്ത് അൾട്രാസൗണ്ട് ട്രാൻസ്ഡ്യൂസർഘടിപ്പിച്ച വഴക്കമുള്ള ഒരു കുഴൽ (നിരീക്ഷണഉപകരണം) ഉണ്ടാകും.

അനസ്തെറ്റിസ്റ്റ് ആ നിരീക്ഷണ ഉപകരണം താങ്കളുടെ തൊണ്ടയിലൂടെ ഈസോഫാഗസിലേയ്ക്ക് (താങ്കളുടെ വായിലൂടെ വയറ്റിലേയ്ക്കുള്ള വഴി) കടത്തും. താങ്കൾ മയക്കത്തിലായിരിക്കുമ്പോഴാണ് ഇത് ചെയ്യുന്നത് എന്നതിനാൽ അസ്വസ്ഥതയൊന്നും ഉണ്ടാകില്ല. ഈസോഫാഗസ് ഹൃദയത്തിന് നേരെ പുറകിലാണെന്നതിനാൽ ഡോക്ടർക്ക് താങ്കളുടെ ഹൃദയത്തിന്റെ കൂടുതൽ വിശദമായ ചിത്രങ്ങൾ ലഭിക്കാൻ ഈ സമീപനം സഹായകരമാകും.

ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫിയുടെ അപായങ്ങളും പാർശ്വഫലങ്ങളുമെന്തെല്ലാം?

ഹൃദയശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന രോഗികളിൽ പതിവായി ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫി ഉപയോഗിക്കുന്നതും അതിലടങ്ങിയ അപായം നിസ്സാരവുമാണ്. ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫിയുടെ 2013ലെ അമേരിക്കൻ സൊസൈറ്റി ഓഫ് ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫിയുടെയും സൊസൈറ്റി ഓഫ് കാഡിയോവാസ്ക്കുലാർ അനസ്തീഷ്യോളജിസ്റ്റിന്റെയും മാർഗ്ഗനിർദ്ദേശങ്ങൾ പ്രകാരം ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫി വഴി ഉണ്ടാകാനിടയുള്ള സങ്കീർണ്ണതകൾ വായിൽനിന്നുള്ള ലഘുവായ രക്തസ്രാവം (0.01%) ഗുരുതരമായ രക്തസ്രാവം (0.03% മുതൽ 0.8%വരെ), എൻഡോട്രാക്കിയൽകുഴലിന്റെ സ്ഥാന വ്യതിയാനം (0.03%), പല്ലിനുണ്ടാകുന്ന പരുക്ക് (0.03%), കടുത്ത തൊണ്ടവേദന (0.1%) ഈസോഫാഗസിൽ തുളവീഴുക (0 മുതൽ 0.3%) എന്നിവയാണ്.

ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫിയുടെ ഉപയോഗവുമായി ബന്ധപ്പെട്ടവയാണ് ഈ അപായങ്ങൾ, പഠനത്തിന്റെ പ്രത്യേകതയോ ബന്ധപ്പെട്ടതോ അല്ല. താങ്കൾ ഈ പഠനത്തിൽ പങ്കെടുത്താലും ഇല്ലെങ്കിലും നിരീക്ഷണത്തിനും ചികിത്സയ്ക്കുമായി ഹൃദയശസ്ത്രക്രിയാ നടപടിയിൽ ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫി ചെയ്യും.

എന്താണ് ഗ്ലോബൽ ലോങ്കിട്യൂഡിനൽ സ്ട്രെയിൻ?
പതിവ് ട്രാൻസ്-ഇസോഫേജിയൽ എക്കോകാർഡിയോഗ്രഫി ചെയ്യുമ്പോൾ ശേഖരിക്കുന്ന ചിത്രങ്ങൾ അതിനുശേഷം കമ്പ്യൂട്ടറിൽ വച്ച് ഹൃദയ പ്രവർത്തനത്തിന്റെ അളവ് വസ്തുനിഷ്ഠമായി നിർണ്ണയിക്കുന്ന പുതിയ സങ്കേതമാണ് ഗ്ലോബൽ ലോങ്കിട്യൂഡിനൽ സ്ട്രെയിൻ.

ഞങ്ങൾ എന്തുകൊണ്ട് ഈ പഠനം നടത്തുന്നു?

സിവോഫ്ലൂറൈൻ അല്ലെങ്കിൽ പ്രൊപ്പൊഫോളുപയോഗിച്ച് മയക്കൽ നടത്തിയ ഗുരുതരമായ അയോർട്ടിക് സ്റ്റെനോസിസുള്ള വാൽവ് മാറ്റിവയ്ക്കൽ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന രോഗികളിലെ ഹൃദയ പ്രവർത്തനത്തിന്റെ സൂക്ഷ്മവും വസ്തുനിഷ്ഠവുമായ എക്കോകാർഡിയോഗ്രഫി പരമായ വിലയിരുത്തലും ഗ്ലോബൽ ലോങ്കിട്യൂഡിനൽ സ്ട്രെയിനുമായുള്ള താരതമ്യവും നടത്താനാണ് ഈ പഠനം ഉദ്ദേശിക്കുന്നത്. ഗുരുതരമായ അയോർട്ടിക് സ്റ്റെനോസിസ് ഉള്ള രോഗികളിൽ കുറഞ്ഞ ഗ്ലോബൽ ലോങ്കിട്യൂഡിനൽ സ്ട്രെയിനുള്ള അപായഘടകങ്ങൾ മനസ്സിലാക്കാനും ഇത് സഹായിക്കും.

പഠനമാരംഭിച്ചശേഷം താങ്കൾക്ക് പിൻമാറാനാകുമോ?
താങ്കളുടെ പഠനത്തിലുള്ള പങ്കാളിത്തം പൂർണമായും സ്വമേധയാ ഉള്ളതും പഠനത്തിൽ നിന്ന് പിൻമാറാൻ സ്വാതന്ത്ര്യം ഉള്ളതും ആണ്. അങ്ങനെ ചെയ്യുന്നതുകൊണ്ട് ഈ ആശുപത്രിയിലെ താങ്കളുടെ സാധാരണ ചികിത്സയെ ഒരുവിധത്തിലും ബാധിക്കുകയില്ല.

പഠനസംബന്ധിയായി എന്തെങ്കിലും പരുക്കുണ്ടായാൽ എന്തുചെയ്യും?
താങ്കൾ ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ട് താങ്കൾക്ക് ഒരു പരിക്കും ഉണ്ടാവുമെന്ന് ഞങ്ങൾ പ്രതീക്ഷിക്കുന്നില്ല, പക്ഷേ താങ്കൾക്ക് പാർശ്വഫലങ്ങളോ പ്രശ്നങ്ങളോ ഉണ്ടാവുകയാണെങ്കിൽ ആ സങ്കീർണ്ണതകൾ താങ്കൾക്ക് അധികച്ചിലവുണ്ടാകാതെ ഞങ്ങൾ ചികിത്സിക്കും

പഠനത്തിന് താങ്കൾ അധികം പണം നൽകണോ?

വേണ്ട

താങ്കളുടെ വ്യക്തിപരമായ വിവരങ്ങൾ രഹസ്യമായി വയ്ക്കുമോ ?
താങ്കളുടെ വ്യക്തിവിവരങ്ങൾ രഹസ്യമായി സൂക്ഷിക്കും. പഠനഫലങ്ങൾ ഒരു വൈദ്യശാസ്ത്ര ജേർണലിൽ പ്രസിദ്ധീകരിക്കുമെങ്കിലും താങ്കളുടെ പേരു വിവരങ്ങൾ പ്രസിദ്ധീകരണത്തിലോ പഠനഫലങ്ങളുടെ പ്രദർശനത്തിലോ ഉണ്ടാവില്ല.
താങ്കൾക്ക് കൂടുതൽ എന്തെങ്കിലും ചോദ്യങ്ങൾ ഉണ്ടെങ്കിൽ ദയവായി ഡോക്ടറോട് ചോദിക്കുക
ഡോ. ശ്രീനിവാസ് വി ഗാധിംഗ്ലജ്കർ, പ്രൊഫസർ, ഡിപ്പാർട്ട്മെന്റ് ഓഫ് അനസ്തീഷ്യോളജി
ഫോൺ 9446304043

പ്രധാന ഗവേഷകൻ ഡോ. മാർക്കോസ് എൽ പരേറ്റ്, സീനിയർ റെസിഡന്റ്, ഡിപ്പാർട്ട്മെന്റ് ഓഫ് അനസ്തീഷ്യോളജി (ഫോൺ -8853303722. ഇമെയിൽ. paretmarcos@gmail.com

പഠനത്തിന്റെ നൈതീക അനുവാദസംബന്ധമായ വിശദീകരണങ്ങൾക്ക് **SCTIMST** നൈതീക കമ്മിറ്റി മെംബർ സെക്രട്ടറി യെ ബന്ധപ്പെടാൻ
Dr Srinivas G
ഫോൺ- 0484 - 2524689
ഇമെയിൽ -iec.mem.sec@sctimst.ac.in

പ്രധാന ഗവേഷകന്റെ പേരും ഒപ്പും

ഡോ. മാർക്കോസ് എൽ പരേറ്റ്,

ഫോൺ -8853303722

പ്രസ്ഥാവനയും സമ്മതപത്രവും

ഞാൻ..... വയസ്സ് (വർഷത്തിലും മാസത്തിലും) .....
.(അച്ഛൻ/അമ്മയുടെ പേര്)..... .

അയോർട്ടിക് വാൽവ് മാറ്റിവയ്ക്കൽ ശസ്ത്രക്രിയയ്ക്ക് വിധേയരാകുന്ന ഗുരുതരമായ അയോർട്ടിക് സ്റ്റെനോസിസ് രോഗികളുടെ സ്ട്രെയിനിന്റെ അളവ് നിർണ്ണയത്തിൽ സിവോഫ്ളൂറൈന്റെയും പ്രൊപ്പൊഫോളിന്റെയും പ്രഭാവം തമ്മിലുള്ള താരതമ്യം എന്ന പഠനത്തിൽ പങ്കെടുക്കാവാൻ സമ്മതം നൽകുന്നു.

(കോളങ്ങൾ അടയാളപ്പെടുത്തുക).

- പഠനസംബന്ധിയായി എനിക്കു നൽകിയ തിയതിയിലെ..... വിവരങ്ങൾ വായിച്ചു എന്നും എനിക്കുണ്ടായ സംശയങ്ങൾ പരിഹരിക്കാൻ അവസരം ലഭിച്ചു എന്നും ഉറപ്പാക്കുന്നു.
- എന്റെ ഈ പഠനത്തിലുള്ള പങ്കാളിത്തം പൂർണ്ണമായും സ്വമേധയാ ആണെന്നും അനുവാദം എനിക്ക് ഏതുസമയത്തും എന്റെ ചികിത്സയെ ബാധിക്കാതെ പിൻവലിക്കാൻ അവകാശമുണ്ടെന്നും ഞാൻ മനസ്സിലാക്കുന്നു. [ ]
- ഞാൻ ഈ പഠനത്തിൽ നിന്നും പിൻമാറിയാലും പഠനം നടത്തുന്നവർക്കും സ്ഥാപനത്തിലെ നൈതിക കമ്മിറ്റി അംഗങ്ങൾക്കും നിയന്ത്രണാധികാരികൾക്കും എന്റെ ആരോഗ്യരേഖകൾ പരിശോധിക്കുന്നതിന് എന്റെ അനുവാദം ആവശ്യമില്ലെന്ന് ഞാൻ മനസ്സിലാക്കുന്നു. അതിനോട് ഞാൻ യോജിക്കുന്നു.[ ]
- എന്നെ തിരിച്ചറിയാനുതകുന്ന വിവരങ്ങൾ ഒന്നും മറ്റുള്ളവർക്കു നൽകുകയോ പ്രസിദ്ധീകരിക്കുകയോ ചെയ്യില്ലെന്ന് ഞാൻ മനസ്സിലാക്കുന്നു. [ ]
- ശാസ്ത്രീയ ആവശ്യങ്ങൾക്കാണെങ്കിൽ ഈ പഠനത്തിലെ വിവരങ്ങൾ ഉപയോഗിക്കുന്നതിന് ഞാൻ പരിധി കൽപ്പിക്കുന്നില്ല[ ]
- എനിക്ക് പഠനത്തെപ്പറ്റിയോ പങ്കാളിയുടെ അവകാശങ്ങളെപ്പറ്റിയോ കൂടുതൽ അറിയണമെങ്കിൽ പ്രധാനഗവേഷകയെ ബന്ധപ്പെടാനുള്ള നമ്പർ നൽകിയിട്ടുണ്ട് [ ]
- ഞാൻ സ്വമേധയാ പഠനത്തിൽ പങ്കെടുക്കാൻ സമ്മതിക്കുന്നു [ ]
- സമ്മതപത്രത്തിന്റെ ഒപ്പിട്ട ഒരു കോപ്പി എനിക്കു കിട്ടി [ ]

പേര്

ഒപ്പ്

തീയതി

സാക്ഷിയുടെ പേര്

പങ്കെടുക്കുന്ന ആളുമായുള്ള ബന്ധം

തീയതി

(സമ്മതം വാങ്ങുന്നയാൾ)

മെഡിക്കൽ റിസർച്ച് പ്രോജക്ടിനാവശ്യമായ സമ്മതപത്രത്തിനു വേണ്ടുന്ന എല്ലാ ഘടകങ്ങളും തൃപ്തികരമായി നിർവഹിച്ചിരിക്കുന്നുവെന്ന് ഞാൻ ബോധ്യപ്പെടുത്തുന്നു. പഠനപങ്കാളിയുമായി ഗവേഷണപദ്ധതിയെപ്പറ്റി സാങ്കേതികേതര പദങ്ങളുപയോഗിച്ച് എല്ലാ വിവരങ്ങളെപ്പറ്റിയും ചർച്ച നടത്തുകയും പ്രതീക്ഷിക്കാവുന്ന അപകടസാധ്യതകളും പാർശ്വഫലങ്ങളും വിശദീകരിക്കുകയും ചെയ്തു. പങ്കാളിയെ ചോദ്യങ്ങൾ ചോദിക്കാൻ പ്രേരിപ്പിക്കുകയും എല്ലാ ചോദ്യങ്ങൾക്കും ഉത്തരം നൽകുകയും ചെയ്തു എന്നും ഞാൻ സാക്ഷ്യപ്പെടുത്തുന്നു.

സമ്മതപത്രം വാങ്ങുന്ന ആളുടെ പേരും ഒപ്പും

ഡോ. മാർക്കോസ് എൽ പരേറ്റ്,

ഫോൺ -8853303722

സാക്ഷി

MASTER CHARTS

Sl no	HOSPITAL NO	Study Group	AGE	SEX	BSA	PREOPERATIVE DETAILS	HYPERTENSION	DIABETES MELLITUS	CCB USE
1	488631	Sevoflurane	62	Female	1.60		YES	NO	NO
2	486488	Sevoflurane	65	Female	1.74		NO	NO	NO
3	487979	Sevoflurane	45	Female	2.17		YES	YES	NO
4	487797	Sevoflurane	61	Female	1.50		NO	NO	NO
5	496622	Sevoflurane	65	Female	1.64		YES	NO	YES
6	498595	Sevoflurane	51	Female	1.76		YES	YES	NO
7	500481	Sevoflurane	73	Female	1.80		NO	NO	NO
8	504832	Sevoflurane	73	Female	1.94		YES	YES	NO
9	501965	Sevoflurane	74	Female	2.20		NO	YES	NO
10	501728	Sevoflurane	67	Female	1.63		NO	NO	NO
11	500264	Sevoflurane	64	Female	1.98		YES	YES	YES
12	498419	Sevoflurane	54	Female	1.80		YES	NO	YES
13	500077	Sevoflurane	55	Female	1.50		NO	YES	NO
14	495973	Sevoflurane	50	Female	1.53		NO	YES	NO
15	501404	Sevoflurane	57	Female	1.30		NO	NO	NO
16	504660	Sevoflurane	62	Female	1.82		YES	NO	YES
17	503423	Sevoflurane	76	Female	1.63		YES	NO	YES
18	510567	Sevoflurane	67	Female	2.20		YES	YES	NO
19	507182	Sevoflurane	58	Female	1.60		YES	YES	NO
20	513506	Sevoflurane	58	Female	1.67		NO	NO	NO
21	501545	Sevoflurane	47	Female	1.71		YES	YES	NO
22	513345	Sevoflurane	69	Female	1.62		YES	NO	NO
23	457392	Propofol	59	Female	1.90		YES	YES	YES
24	320424	Propofol	66	Female	1.70		NO	NO	NO
25	414026	Propofol	65	Female	1.85		NO	NO	NO
26	489186	Propofol	60	Female	1.64		NO	NO	NO
27	380671	Propofol	60	Female	1.28		NO	NO	NO
28	430217	Propofol	67	Female	1.69		YES	YES	YES
29	466931	Propofol	65	Female	1.61		YES	NO	YES
30	502632	Propofol	57	Female	1.68		NO	NO	NO
31	500926	Propofol	57	Female	1.75		NO	YES	NO
32	502809	Propofol	62	Female	1.50		YES	YES	YES
33	492487	Propofol	64	Female	1.66		NO	YES	NO
34	503853	Propofol	60	Female	1.88		NO	YES	NO
35	486889	Propofol	61	Female	2.00		YES	YES	NO
36	488828	Propofol	60	Female	1.70		NO	NO	NO
37	293724	Propofol	50	Female	1.50		NO	NO	NO
38	491544	Propofol	66	Female	1.63		YES	NO	YES
39	495551	Propofol	60	Female	1.63		NO	NO	NO
40	497644	Propofol	64	Female	1.69		NO	NO	NO
41	475487	Propofol	64	Female	1.65		YES	NO	NO
42	424981	Propofol	64	Female	1.77		YES	YES	NO
43	512811	Propofol	63	Female	1.30		NO	NO	NO

ARB/ ACE INHIBITOR USE	BETA BLOCKER USE	SEPTAL WALL THICKNESS SYSTOLE (mm)	SEPTAL WALL THICKNESS DIASTOLE (mm)	INFEROLATERAL WALL THICKNESS SYSTOLE (mm)	INFEROLATERAL WALL THICKNESS DIASTOLE (mm)	AORTIC VALVE MEAN GRADIENT (mm HG)
NO	NO	24	20	22	20	58
NO	NO	25	24	24	20	58
NO	YES	18	17	19	16	76
NO	NO	19	15	17	15	58
NO	NO	16	12	16	12	58
NO	YES	13	10	16	13	68
NO	NO	14	12	9	9	40
NO	YES	28	20	19	17	59
NO	NO	16	14	15	13	48
NO	NO	16	13	17	14	70
YES	NO	19	15	17	11	44
YES	NO	13	12	14	12	44
NO	NO	15	13	12	10	40
NO	NO	13	12	14	12	47
NO	NO	12	10	12	10	54
YES	YES	19	15	20	16	70
NO	NO	16	15	14	9	44
YES	YES	16	13	15	13	38
YES	NO	17	15	16	13	63
NO	NO	15	12	14	13	72
YES	NO	16	11	12	8	36
NO	YES	16	12	15	12	46
NO	NO	18	13	16	12	45
YES	NO	13	12	13	12	49
NO	NO	13	12	15	12	65
NO	NO	16	11	14	11	46
NO	NO	13	12	13	11	50
NO	NO	14	11	16	12	46
NO	NO	17	13	15	10	85
NO	NO	11	9	10	9	41
NO	NO	18	12	12	10	57
NO	NO	13	9	11	10	47
NO	NO	16	13	12	10	52
NO	NO	11	10	16	9	58
YES	YES	22	18	20	18	54
NO	NO	11	9	12	9	43
NO	NO	15	9	16	10	41
NO	NO	16	13	15	13	71
NO	NO	17	15	16	11	57
NO	NO	24	19	21	15	70
YES	NO	15	14	15	14	47
YES	NO	21	18	23	14	44
NO	YES	9	7	9	7	63

AORTIC VALVE PEAK VELOCITY (m/s)	PRE-OPERATIVE EF (%)	PRE CPB-DATA	PRE CPB HR	SBP	DBP	MAP	PP
4.98	60		60	120	60	80	60
4.30	50		51	160	70	90	90
4.00	55		60	120	70	87	50
5.10	72		92	103	70	81	33
4.60	60		70	146	82	103	64
4.50	78		84	123	74	90	49
4.00	75		68	108	64	79	44
4.90	60		62	110	50	70	60
4.20	68		50	100	58	72	42
4.90	73		64	110	60	76	50
4.10	77		80	130	70	90	60
4.70	59		92	130	82	98	48
3.60	50		52	110	48	68	62
5.10	51		62	110	60	76	50
4.80	53		110	110	70	83	40
5.70	63		60	144	75	98	69
4.20	62		64	130	60	83	70
4.00	60		50	140	70	93	70
5.20	56		68	126	68	88	58
5.20	60		90	140	90	105	50
3.80	80		74	140	90	106	50
4.20	64		80	126	86	99	40
4.00	76		90	116	86	96	30
4.30	68		65	116	76	86	30
5.20	64		90	107	84	90	23
3.40	69		90	120	70	86	50
3.98	77		65	116	78	91	38
4.20	76		62	138	72	94	66
5.60	56		70	140	80	93	40
4.10	67		80	124	60	81	64
4.70	69		64	120	72	64	48
4.50	61		84	131	71	83	60
4.10	71		60	100	66	34	77
4.80	56		74	150	76	101	74
4.30	66		62	120	70	86	50
4.20	63		70	116	78	91	38
3.80	64		62	120	70	85	50
5.20	68		64	140	56	102	84
4.70	65		60	130	70	83	60
5.40	63		76	113	58	75	55
5.00	65		68	140	90	106	50
4.50	64		70	156	72	100	84
5.00	59		80	110	70	90	60

CVP	LVEDA (cm2)	LVOT CSA (cm2)	LVOT VTI (cm)	LVOT SV (ml)	LVOT SV index (ml/m2)	LVOT CO (L/min)
8	12.0	3.20	18.0	57.0	35.60	3.40
13	13.0	4.00	19.0	76.0	43.60	3.87
12	13.0	4.15	23.0	95.0	43.90	5.60
7	11.0	3.80	17.0	64.0	42.60	5.80
9	16.0	3.10	15.0	46.0	28.30	3.20
8	15.0	2.90	16.0	46.0	27.20	3.80
12	15.0	3.14	16.0	49.0	27.20	3.30
12	10.0	2.85	17.5	50.0	25.77	3.10
13	16.8	4.15	18.9	78.0	35.40	3.90
7	18.0	4.50	19.0	85.0	53.00	5.40
8	16.5	4.00	18.0	70.0	35.30	5.00
7	12.0	3.20	17.0	54.0	30.00	4.90
10	12.0	4.15	19.4	84.0	56.00	4.40
7	12.0	3.00	18.0	54.0	34.80	3.30
8	13.0	2.50	15.0	38.0	26.30	4.10
11	12.0	3.10	17.0	52.0	28.80	3.10
9	15.0	3.20	15.0	48.0	29.40	3.00
9	15.0	3.00	23.9	72.0	32.70	3.60
8	15.0	3.50	16.0	56.0	35.00	3.80
10	53.0	3.10	11.6	36.0	21.50	3.20
10	12.0	3.00	12.0	36.0	21.10	2.60
10	14.0	3.20	17.0	55.0	33.90	4.40
11	14.0	3.80	17.4	66.0	34.70	5.90
7	15.0	4.30	17.0	73.0	42.90	4.70
5	10.0	3.10	18.0	56.0	30.20	5.00
6	12.0	3.90	15.0	58.0	35.00	5.30
7	14.0	2.90	18.0	52.0	40.60	3.30
9	14.0	2.80	22.0	56.0	31.10	3.40
5	14.0	3.30	15.0	49.0	30.60	3.40
13	16.0	3.70	18.0	66.0	39.20	5.20
11	13.0	3.10	22.0	68.0	38.80	4.30
7	15.0	3.80	16.0	60.0	40.00	5.00
12	15.0	3.20	21.0	67.0	40.36	4.00
9	14.0	3.40	18.0	61.2	32.50	4.50
6	14.0	2.90	22.0	64.0	32.00	3.90
11	13.0	3.40	19.0	64.0	37.60	4.40
10	14.0	3.20	20.0	64.0	42.60	3.90
11	16.0	3.00	19.0	57.0	34.90	3.60
7	15.0	3.30	20.0	66.0	40.40	4.00
12	13.0	2.90	23.0	67.0	39.60	5.00
10	8.0	3.10	15.0	45.0	28.00	3.10
8	18.0	3.14	20.0	63.0	37.00	4.40
8	11.0	3.14	18.0	56.0	43.00	4.50

Aortic valve mean gradient (mm Hg) under GA	SVR (dynes/sec/cm-5	Systemic arterial compliance	Valvuloarterial impedance	SEPTAL WALL THICKNESS (mm)	INFEROLATERAL WALL THICKNESS (mm)	LEFT VENTRICULAR INTERNAL DIAMETER (mm)	LV LONG AXIS LENGTH (mm)
45	1695	0.590	4.60	20.0	20.0	40.0	82.0
40	1584	0.480	2.98	22.0	20.0	38.0	92.0
50	1071	0.870	3.87	17.0	20.0	40.0	85.0
42	1020	1.290	3.40	16.0	15.0	32.0	75.0
53	2312	0.440	7.00	13.0	14.0	35.0	82.0
48	1726	0.550	6.28	13.0	14.0	38.0	87.0
40	1624	0.610	5.40	11.5	13.2	41.3	81.0
59	1200	0.420	6.50	14.2	13.0	28.6	90.0
48	1242	0.840	4.17	14.0	13.0	35.0	88.0
50	1022	1.060	3.01	16.4	15.8	47.7	60.3
44	1312	0.580	5.00	13.5	10.6	40.0	95.0
44	2008	0.620	5.80	13.0	12.0	35.0	83.0
28	1048	0.900	2.46	15.0	13.0	51.7	93.0
38	1672	0.690	4.25	12.0	13.0	35.0	88.0
40	1097	0.650	5.70	13.8	12.4	34.0	92.0
50	2245	0.410	6.70	15.0	16.0	38.0	94.0
35	1973	0.420	5.61	15.0	14.0	38.0	80.0
30	1866	0.460	5.19	13.0	13.0	39.0	78.0
50	1684	0.600	5.00	15.0	13.0	34.0	84.0
50	1788	0.430	8.80	12.8	10.8	46.7	81.0
30	2923	0.420	8.05	15.0	11.0	40.0	80.0
40	1618	0.840	4.89	8.4	12.6	37.0	70.0
30	1152	1.150	3.63	13.2	13.8	42.0	65.9
40	1344	1.430	2.95	13.0	13.0	39.0	85.0
52	1360	1.310	4.70	13.5	16.0	38.0	86.0
30	1733	0.710	4.21	14.0	14.0	35.0	78.0
44	2036	1.060	3.94	14.0	13.0	32.0	74.0
32	2047	0.470	5.46	13.0	13.0	47.0	89.0
46	2070	0.760	6.07	13.4	11.0	40.0	90.0
28	1046	0.610	3.87	13.0	13.0	36.0	89.0
43	986	0.800	4.20	13.0	13.0	45.0	92.0
41	1216	0.660	4.30	14.0	14.0	45.0	93.0
38	1300	1.340	3.41	14.0	14.0	42.0	94.0
41	1475	0.430	5.80	10.0	9.0	40.0	95.0
36	1682	0.640	4.80	19.0	18.0	43.0	91.0
30	1454	0.980	3.88	9.0	9.0	46.0	94.0
28	1538	0.850	2.65	12.0	12.0	43.0	93.0
54	2022	0.410	4.46	13.8	14.0	46.0	93.0
41	1540	0.670	4.27	14.0	12.0	42.0	95.0
54	1008	0.720	4.21	14.3	16.3	36.8	87.0
40	2477	0.560	6.42	13.0	14.0	36.0	7.0
29	1672	0.440	3.48	12.0	14.0	41.0	76.0
59	1440	0.710	3.46	12.0	10.0	37.0	65.0

LV MASS (gm)	LV MASS INDEX (gm/m2)	E/e'	RELATIVE WALL THICKNESS	PRE CPB GLS	2 CHAMBER STRAIN	BASAL ANTERIOR	MID ANTERIOR	APICAL ANTERIOR	BASAL INFERIOR
240	150.0	16.0	1.00	-13	-18	-15	-19	-25	-11
270	155.0	15.0	1.05	-12	-10	-8	-6	-8	-12
409	185.0	11.0	0.47	-11	-9	-11	-10	-8	-7
252	168.0	8.0	0.93	-9	-10	-12	-9	-8	-10
260	158.0	10.0	0.80	-8	-8	-7	-10	-8	-6
201	114.0	7.0	0.73	-14	-12	-10	-13	-12	-11
196	108.0	11.0	0.64	-13	-15	-16	-14	-27	-22
245	128.0	7.0	0.92	-7	-7	-3	-7	-7	-8
242	110.0	7.0	0.74	-14	-16	-14	-6	-19	-9
254	155.0	9.0	0.66	-7	-9	-6	-7	-11	6
228	115.0	11.0	0.53	-9	-5	-7	6	-10	-5
278	154.0	13.0	0.68	-10	-11	-10	-13	-9	-14
274	182.0	10.0	0.50	-7	-6	-3	-7	-6	-7
266	173.0	15.0	0.74	-11	-12	-10	-14	-13	-15
282	216.0	10.0	0.70	-13	-15	-15	-17	-13	-12
255	140.0	7.0	0.84	-8	-10	-9	-11	-10	-12
293	179.0	11.0	0.73	-12	-14	-12	-14	-16	-18
253	115.0	6.0	0.66	-8	-8	-6	-9	-9	-8
197	123.0	15.0	0.76	-11	-14	-13	-14	-18	-12
188	112.0	9.0	0.46	-8	-7	-6	-4	-11	-7
200	117.0	6.0	0.55	-10	-12	3	-19	-15	-14
120	73.6	9.0	0.68	-14	-22	-7	-14	-39	-34
247	130.0	9.0	0.62	-14	-26	-19	-18	-41	-23
201	125.0	9.0	0.66	-12	-14	-24	-10	-18	-9
210	114.0	8.0	0.84	-14	-13	-16	-10	-11	-15
249	152.0	13.0	0.80	-15	-18	-18	-16	-10	-19
223	174.0	14.0	0.68	-9	-11	-12	-9	-8	-13
206	122.0	12.2	0.55	-12	-11	-2	-15	-15	-13
260	162.0	16.0	0.55	-15	-18	-16	-20	-18	-15
174	104.0	14.0	0.72	-10	-9	-7	-12	-13	-7
236	134.0	10.0	0.57	-15	-20	-12	-9	-27	-7
190	127.0	13.0	0.66	-15	-17	-15	-19	-17	-12
280	175.0	17.0	0.57	-10	-12	-10	-11	-13	-15
241	127.0	15.0	0.45	-13	-15	-16	-14	-12	-13
238	119.0	16.0	0.83	-12	-14	-11	-15	-13	-14
230	135.0	16.0	0.39	-16	-16	-17	-14	-12	-16
220	146.0	8.0	0.55	-14	-16	-18	-15	-19	-14
280	175.0	13.0	0.60	-10	-9	-9	-7	-8	-10
240	150.0	10.0	0.57	-14	-17	-18	-15	-17	-14
253	149.0	8.0	0.88	-13	-17	-13	-14	-21	-18
240	145.0	10.0	0.77	-7	-5	-3	-8	-10	-7
211	119.0	11.0	0.68	-11	-9	-10	-11	-9	-17
258	198.0	5.5	0.54	-12	-17	-10	-8	-26	-18

MID INFERIOR	APICAL INFERIOR	APEX	3CHAMBER STRAIN	BASAL ANTEROSEPTAL	MID ANTEROSEPTAL	APICAL ANTEROSEPTAL	BASAL INFEROLATERAL
-9	-26	-25	-10	-9	-10	-11	-12
-16	-10	-10	-12	-12	-16	-10	-14
-3	-8	-9	-13	-9	-12	-14	-15
-13	-10	-8	-8	-7	-6	-9	-11
-7	-8	-10	-9	-10	-8	-5	-12
-14	-15	-9	-15	-14	-16	-12	-17
-14	-18	-23	-11	3	-8	-18	-6
-8	-9	-8	-8	-5	-11	-7	-8
-12	-31	-25	-13	-9	3	-26	-14
-19	-15	-13	-8	-20	7	-8	10
-8	-6	-8	-8	-3	-11	-12	-8
-15	-11	-5	-9	-10	-11	-8	-8
-5	-8	-6	-8	-7	-11	-9	-3
-11	-10	-11	-10	-12	-15	-8	-8
-17	-15	-16	-12	-9	-14	-16	-8
-7	-9	-12	-7	-6	-8	-9	-9
-10	-18	-10	-12	-11	-13	-10	-14
-8	-9	-9	-10	-10	-8	-11	-8
-16	-12	-13	-9	-11	-8	-6	-8
-6	-13	-9	-8	-2	4	-17	-14
-16	-14	-12	-11	-14	-18	-6	-20
-15	-25	-32	-14	-5	-17	-17	-16
-16	-32	-36	-9	-7	-22	6	-13
-9	-15	-13	-10	-10	-9	-12	-13
-10	-14	-15	-17	-19	-21	-15	-13
-18	-24	-21	-12	-16	-12	-10	-9
-11	-10	-14	-8	-6	-5	-8	-10
-11	-7	-14	-11	-6	-17	-17	3
-22	-18	-18	-12	-13	-11	-9	-16
-6	-9	-12	-12	-11	-13	-9	-12
-21	-30	-29	-9	-6	-15	-11	-7
-19	-21	-16	-13	-15	-17	-11	-13
-10	-12	-13	-9	-11	-7	-12	-9
-17	-15	-18	-13	-11	-14	-13	-12
-10	-17	-18	-9	-10	-7	-9	-11
-17	-18	-18	-19	-17	-19	-21	-23
-16	-15	-15	-13	-11	-17	-13	-15
-11	-10	-8	-12	-10	-11	-13	-9
-19	-17	-19	-11	-12	-10	-11	-13
-8	-27	-23	-8	-4	-28	-8	9
8	-12	-11	-6	6	5	-23	-12
-12	-9	-8	-10	14	-10	-18	-16
-9	-22	-24	-10	2	-7	-20	-13

MID INFEROLATERAL	APICAL INFEROLATERAL	APEX	4CHAMBER STRAIN	BASAL ANTEROLATERAL	MID ANTEROLATERAL
-11	-9	-10	-9	-9	-18
-12	-10	-8	-14	-8	-16
-13	-12	-16	-11	-13	-11
-8	-6	-9	-9	-11	-10
-13	-12	-12	-7	-6	-8
-14	-17	-15	-15	-17	-13
-13	-17	-18	-12	-6	-12
-8	-15	-10	-6	-11	-10
-5	-17	-21	-13	-6	-15
-17	-20	-14	-4	-14	10
-11	-5	-6	-15	-14	-19
-7	-11	-8	-10	-12	-7
-7	-9	-10	-8	-10	-7
-12	-7	-8	-11	-12	-14
-7	-12	-18	-12	-15	-10
-7	-5	-5	-7	-6	-8
-9	-14	-13	-10	-7	-9
-7	-13	-13	-9	-7	-9
-11	-12	-7	-10	-7	-12
10	-11	-13	-9	-8	2
-14	-7	-6	-9	-13	5
-17	-12	-15	-8	-6	-7
-12	-7	-5	-9	-10	-5
-8	-5	-13	-12	-13	-11
-10	-22	-19	-12	-20	-15
-14	-15	-8	-15	-20	-15
-7	-8	-12	-8	-7	-11
-13	-13	-15	-13	-3	-14
-14	-12	-9	-15	-16	-17
-15	-11	-13	-9	-8	-11
-7	-16	-13	-18	-15	-11
-10	-14	-11	-15	-10	-17
-8	-10	-6	-9	-10	-8
-10	-15	-16	-17	-20	-15
-12	-9	-5	-13	-11	-14
-16	-19	-18	-13	-15	-11
-13	-12	-10	-13	-11	-17
-13	-12	-16	-9	-7	-10
-10	-13	-8	-14	-15	-13
-13	-14	-11	-16	-10	-9
-8	8	-10	-9	-3	-22
-15	-11	-14	-13	-7	-11
-8	-9	-14	-9	-9	8

APICAL ANTEROLATERAL	BASAL INFEROSEPTAL	MID INFEROSEPTAL	APICAL INFEROSEPTAL	APEX
-3	-15	7	-16	-9
-10	-16	-14	-12	-8
-10	-9	-14	-13	-7
-7	-5	-10	-9	-4
-6	-8	-7	-7	-7
-18	-14	-19	-13	-11
-24	-23	-12	-12	-13
-17	-11	-6	10	-16
-20	-9	-13	-12	-16
-5	-10	13	-6	-5
-23	-6	5	-30	-26
-8	-10	-13	-11	-9
-10	-6	-9	-12	-14
-9	-8	-13	-7	-14
-12	-13	-15	-10	-9
-9	-7	-10	-5	-4
-13	-12	-11	-10	-11
-9	-11	-9	-10	-8
-13	-10	-9	-11	-8
-19	-6	-4	-12	-15
-9	-19	-11	-10	-9
-8	-12	-5	-10	-8
-13	-8	-5	-11	-12
-8	-12	-11	-14	-15
-10	-13	-12	-9	-6
-13	-15	-18	-16	-8
-7	-6	-8	-7	-8
-16	-5	-19	-18	-17
-20	-12	-13	-17	-10
-10	-13	-9	-7	-5
-17	-10	-17	-31	-23
-14	-13	-19	-18	-14
-9	-11	-10	-9	-6
-17	-19	-16	-17	-15
-15	-13	-15	-13	-10
-14	-13	-12	-15	-11
-15	-13	-16	-12	-7
-11	-9	-9	-8	-9
-17	-14	-12	-15	-12
-27	-12	-8	-27	-27
-9	-10	-9	-11	-10
-16	-11	-24	-11	-14
-9	-10	-21	-10	-9

PRE CPB GCS	TG BASAL STRAIN	INFERIOR	INFEROSEPTAL	ANTEROSEPTAL	ANTERIOR	ANTERIOLATERAL
-31	-31	-33	-21	-27	-46	-32
-28	-28	-30	-32	-20	-20	-36
-26	-20	-22	-21	-19	-18	-23
-31	-24	-27	-25	-23	-30	-20
-29	-32	-34	-30	-36	-28	-35
-26	-28	-30	-26	-25	-29	-31
-27	-24	-17	-18	-19	-36	-25
-31	-31	-30	-18	-47	-31	-32
-27	-29	-18	-40	-23	-36	-28
-21	-21	-16	-18	-23	-21	-27
-31	-27	-24	-31	-23	-31	-38
-28	-24	-26	-22	-20	-24	-23
-20	-21	-23	-19	6	-44	-19
-25	-28	-22	-28	-31	-29	-26
-27	-23	-20	-22	-25	-24	-27
-30	-30	-32	-34	-28	-27	-33
-26	-25	-21	-19	-28	-30	-27
-23	-22	-17	-25	-15	-16	-23
-28	-26	-30	-22	-27	-29	-22
-17	-16	-11	-12	-15	-28	-17
-20	-24	-27	16	-29	-24	-33
-24	-18	-18	-22	-22	-22	-23
-29	-26	-25	-19	-28	-31	-23
-28	-26	-27	-27	-20	-30	-28
-22	-20	-27	-20	-19	-21	-17
-19	-19	-17	-21	-17	-25	-22
-27	-29	-27	-27	-30	-26	-29
-25	-28	-20	-28	-34	-31	-37
-24	-22	-26	-25	-22	-27	-20
-25	-24	-28	-21	-28	-21	-20
-26	-30	-31	-18	-17	-42	-35
-26	-25	-30	-26	-28	-28	-30
-28	-27	-29	-30	-26	-25	-28
-21	-20	-24	-20	-19	-26	-19
-27	-25	-30	-20	-27	-29	-25
-22	-20	-23	-17	-20	-18	-22
-24	-21	-18	-26	-25	-21	-19
-26	-24	-28	23	-21	-26	-25
-20	-20	-23	-17	-25	-20	-19
-29	-18	-9	-17	-15	-30	-18
-21	-16	-10	-7	-24	-28	-12
-20	-18	-19	-22	-32	-7	-23
-22	-20	-14	-13	-20	-32	-33

INFEROLATERAL	TG MID STRAIN	INFERIOR	INFEROSEPTAL	ANTEROSEPTAL	ANTERIOR
-27	-28	-49	-19	-25	-43
-30	-36	-40	-32	-28	-42
-17	-32	-36	-34	-26	-30
-19	-38	-36	-38	-41	-34
-29	-28	-25	-30	-32	-27
-27	-22	-19	-24	-26	-21
-28	-26	-31	-17	-27	-40
-31	-29	-19	-35	-57	-37
-33	-28	-27	-22	-41	-33
-21	-19	-13	-18	-24	-38
-24	-32	-37	-27	-35	-41
-29	-32	-34	-30	-28	-36
-26	-26	-28	-18	-22	-32
-32	-23	-24	-26	-21	-20
-20	-28	-27	-28	-27	-32
-26	-28	-27	-25	-32	-32
-25	-28	-26	-30	-21	-32
-16	-25	-27	-24	-23	-28
-26	-30	-32	-30	-31	-29
-22	-13	-4	-14	-7	-30
-19	-21	-22	-19	-17	-32
-9	-25	-20	-25	-28	-28
-29	-35	-35	-30	-38	-41
-24	-30	-35	-28	-27	-29
-16	-22	-27	-20	-21	-26
-29	-26	-28	-24	-28	-21
-34	-25	-28	-26	-23	-24
-27	-23	-35	-12	-42	-19
-12	-26	-29	-25	-24	-25
-26	-23	-21	-23	-27	-23
-32	-24	-16	-21	-16	-43
-25	-25	-20	-23	-27	-25
-24	-28	-29	-27	-25	-31
-12	-22	-25	-25	-19	-23
-19	-29	-29	-30	-27	-30
-20	-22	-26	-20	-18	-21
-21	-28	-30	-29	-25	-29
-21	-29	-30	-31	-28	-29
-16	-23	-26	-23	-21	-25
-25	-32	-28	-45	-30	-35
-18	-20	-20	-8	-13	-42
-17	-16	-15	-12	7	-17
-15	-14	-13	-8	-17	-32

ANTEROLATERAL	INFEROLATERAL	TG APICAL STRAIN	INFERIOR	SEPTAL	ANTERIOR	LATERAL
-17	-25	-35	-37	-36	-49	-19
-36	-38	-20	-18	-22	-26	-14
-38	-38	-26	-21	-23	-29	-31
-40	-39	-30	-33	-27	-28	-32
-31	-23	-27	-28	-23	-29	-31
-23	-19	-28	-30	-25	-31	-26
-13	-27	-30	-25	-38	-37	-27
-32	-22	-33	-40	-43	-31	-25
-21	-33	-24	-25	-30	-24	-20
-14	-10	-23	-17	-24	-23	-27
-39	-23	-33	-35	-30	-38	-30
-32	-32	-28	-26	-30	-28	-28
-28	-25	-6	-9	-5	-8	-4
-28	-19	-26	-28	-25	-23	-28
-25	-29	-30	-34	-28	-27	-30
-28	-24	-32	-36	-35	-28	-27
-26	-33	-22	-21	-23	-25	-19
-22	-26	-22	-21	-25	-20	-22
-28	-30	-30	-34	-26	-28	-32
-15	-23	-25	-26	-23	-26	-24
-20	-25	-14	-15	-18	-9	-20
-25	-28	-28	-21	-28	-27	-33
-29	-41	-30	-39	-50	-10	-42
-33	-28	-28	-31	-27	-26	-28
-17	-21	-24	-28	-20	-22	-26
-30	-25	-27	-28	-28	-25	-31
-28	-21	-27	-29	-27	-30	-22
-21	-27	-25	-29	-33	-21	-20
-27	-26	-24	-28	-20	-24	-16
-19	-25	-30	-32	-31	-27	-30
-20	-31	-25	-16	-10	-37	-22
-27	-18	-25	-30	-26	-21	-23
-30	-26	-29	-31	-27	-29	-27
-24	-22	-21	-20	-18	-26	-20
-28	-30	-27	-29	-27	-26	-26
-23	-24	-24	-28	-23	-25	-20
-27	-28	-23	-25	-23	-20	-24
-27	-29	-25	-21	-29	-30	-20
-23	-20	-20	-20	-17	-21	-20
-30	-34	-39	-34	-36	-43	-41
-20	-20	-32	-30	-37	-36	-28
-22	-34	-33	-44	-37	-37	-16
-7	-10	-32	-31	-33	-30	-43

CPB DETAILS	CLAMP TIME(min)	CPB TIME (min)	VALVE DETAILS	VALVE TYPE	VALVE SIZE
	83	137		INSPIRIS RESILIA	19
	94	158		CHVP	23
	81	134		CHVP	25
	65	98		PM MAGNA	19
	105	139		CHVP	19
	94	157		CHVP	21
	142	213		PM MAGNA	23
	125	180		PM MAGNA	21
	120	183		CHVP	23
	149	179		PM MAGNA	23
	103	144		CHVP	25
	139	240		CHVP	21
	116	179		CHVP	21
	68	116		ON-X	21
	62	92		ON-X	19
	107	168		PM MAGNA	21
	99	154		PM MAGNA	19
	111	168		CHVP	23
	99	140		CHVP	21
	56	81		CHVP TC2	19
	71	104		CHVP	19
	70	96		P MAGNA	19
	83	127		PM Magna	23
	45	60		PM Magna	21
	55	88		ON-X	21
	61	90		CHVP	23
	52	80		PM MAGNA	19
	149	182		PM Magna	21
	50	64		CHVP	19
	111	148		PM MAGNA	19
	63	80		CHVP TC2	21
	112	145		CHVP	17
	96	140		CHVP	19
	111	212		CHVP	21
	60	83		PM Magna	23
	68	90		CHVP	21
	75	105		CHVP	21
	97	132		PM MAGNA	21
	56	75		CHVP	21
	56	123		CHVP	21
	84	103		ON - X	21
	104	150		CHVP	21
	168	208		P MAGNA	19

POST CPB	INOTROPES	VASOPRESSORS	VASODILATORS	VIS while weaning off CPB	POST CPB HB (mg/dl)	LVEDA (cm2)
	No	Noradr 0.025	No	2.5	9.0	13.0
	No	Noradr 0.05	No	5	8.5	14.0
	No	Noradr 0.05	No	5	9.6	10.0
	Adr 0.025	Noradr 0.025	No	5	8.3	12.0
	Adr 0.03	Noradr 0.03	No	6	9.6	14.0
	Adr 0.05	Noradr 0.04	No	9	9.2	16.0
	Adr 0.05	Noradr 0.05	No	10	8.9	17.0
	Adr 0.025	Noradr 0.05	No	7.5	8.6	9.0
	Adr 0.01	Noradr 0.01	No	2	8.8	16.8
	No	No	No	0	12.0	18.0
	No	No	No	0	9.9	20.0
	No	No	No	0	9.6	9.0
	Adr 0.05	Noradr 0.05	No	10	8.8	14.0
	Adr 0.1	Noradr 0.05	No	10	9.3	12.0
	Adr 0.05	Noradr 0.02	No	7	10.2	13.0
	Adr 0.02	Noradr 0.02	No	4	8.6	15.0
	Adr 0.05	Noradr 0.01	No	6	9.0	14.0
	Adr 0.05	No	No	5	11.0	14.0
	No	No	No	0	9.0	15.0
	No	No	No	0	11.0	7.0
	No	No	No	0	9.1	14.0
	No	Noradr 0.05	No	5	9.3	14.0
	No	No	No	0	9.8	15.0
	No	No	No	0	9.9	12.0
	No	No	No	0	10.4	10.0
	Adr 0.05	Noradr 0.05	No	10	10.8	11.0
	Adr 0.05	Noradr 0.05	No	10	11.0	13.0
	No	Noradr 0.02	No	2	8.5	12.0
	Adr0.03	Noradr 0.05	No	8	11.1	13.0
	Dobu 2	Noradr 0.01	No	3	9.7	12.0
	No	No	No	0	10.0	13.0
	Adr 0.05	Noradr 0.025	No	7.5	10.5	14.0
	Adr 0.025	Noradr 0.025	No	5	10.5	12.0
	Adr 0.025	Noradr 0.025	No	5	9.9	15.0
	No	No	No	0	10.3	15.0
	No	No	No	0	10.3	13.0
	Dobu 5	Noradr 0.05	No	10	11.3	13.0
	Adr 0.025	Noradr 0.025	No	5	10.3	14.0
	No	Noradr 0.025	No	2.5	9.5	13.0
	No	No	No	0	11.2	14.0
	No	No	No	0	9.0	8.0
	No	No	No	0	9.5	10.0
	Adr 0.05	Noradr 0.05	No	10	10.6	12.0

HR (per minute)	PACING ?	AORTIC VALVE PROSTHESIS PEAK VELOCITY (m/s)	AORTIC VALVE PROSTHESIS MEAN GRADIENT (mm Hg)	ACCELERATION TIME (ms)	EOAI (cm2/m2)	DVI	ANY PARAVALVULAR LEAK
70	No	2.70	16	85	1.01	0.50	No
65	No	2.60	14	63	0.90	0.60	No
68	No	1.90	11	80	1.24	0.70	No
72	No	3.60	20	100	0.90	0.60	No
68	No	2.00	13	78	0.74	0.30	No
75	No	1.80	12	70	0.70	0.35	No
49	No	1.60	6	66	0.76	0.60	No
62	No	2.90	10	85	0.70	0.60	No
80	No	1.70	11	78	0.75	0.50	No
94	No	2.00	7	92	1.29	0.40	No
82	No	1.25	3	80	2.20	1.30	No
78	No	1.30	8	70	1.00	0.50	No
82	No	3.20	15	75	0.85	0.30	No
60	No	2.80	17	80	0.70	0.30	No
64	No	2.20	10	77	0.80	0.30	No
90	No	1.90	8	80	0.70	0.30	No
80	No	2.20	4	72	0.80	0.40	No
75	No	3.60	15	88	0.82	0.54	No
68	No	1.80	12	72	0.83	0.50	No
90	No	2.00	11	80	0.77	0.50	No
76	No	2.10	15	78	0.80	0.30	No
90	No	2.10	13	64	0.80	0.40	No
90	No	1.50	14	82	0.70	0.50	No
92	No	2.20	7	71	0.40	0.80	No
90	No	2.20	8	60	0.80	0.50	No
82	No	1.60	11	42	0.85	0.60	No
86	No	2.00	15	60	0.85	0.30	No
60	No	2.00	11	90	0.85	0.50	No
90	No	2.20	20	80	0.86	0.30	No
52	No	3.10	16	86	0.80	0.30	No
61	No	1.60	6	70	0.80	0.40	No
60	No	2.40	13	80	1.12	0.50	No
80	No	3.10	17	81	1.35	0.40	No
70	No	2.50	15	74	0.83	0.70	No
70	No	1.80	10	72	0.73	0.40	No
70	No	1.80	10	78	0.89	0.50	No
70	No	1.60	6	88	0.87	0.50	No
78	No	1.60	6	76	0.90	0.50	No
68	No	1.60	10	88	1.50	0.40	No
66	No	2.60	10	48	0.85	0.60	No
80	No	1.80	10	70	0.85	0.30	No
80	No	1.70	7	77	0.72	0.40	No
80	No	2.60	14	72	1.20	0.50	No

LVOT CARDIAC OUTPUT (L/min)	TAPSE (mm Hg)	SVR (dynes/sec/cm-5)	POST CPB GLS	2CHAMBER STRAIN	BASAL ANTERIOR	MID ANTERIOR	APICAL ANTERIOR
3.7	20	1300	-10	-9	-5	-12	-18
4.2	21	900	-11	-14	-10	-16	-12
3.4	17	1170	-12	-10	-9	-10	-12
2.8	16	1500	-8	-8	-7	-8	-7
3.5	20	1600	-10	-11	-11	-12	-10
3.4	16	1100	-14	-13	-11	-16	-12
2.6	18	1500	-20	-28	-20	-22	-47
3.1	17	1200	-12	-9	-9	-17	-8
3.1	20	1100	-15	-14	-13	-16	-10
5.3	20	900	-8	-6	12	-10	-14
5.2	18	980	-8	-5	-6	-10	-11
3.2	19	1100	-11	-10	-12	-9	-10
4.2	15	900	-8	-7	-7	9	-10
3.0	16	1300	-10	-11	-9	-8	-11
2.9	15	1400	-12	-12	-13	-12	-10
3.4	17	1000	-9	-11	-13	-12	-8
3.2	18	1380	-13	-14	-9	-17	-16
5.2	22	1000	-8	-7	-9	-10	-5
3.4	21	980	-10	-10	-13	-7	-10
3.3	20	1100	-5	-3	-3	-12	-3
4.0	19	870	-11	-9	-8	-10	-7
5.2	21	1036	-10	-10	-10	7	-16
3.8	19	1020	-14	-14	-14	-8	-17
5.0	14	1300	-13	-15	-14	-16	-18
3.0	19	900	-13	-10	-8	-13	-9
4.0	17	1360	-16	-15	-13	-20	-19
3.0	20	1100	-10	-7	-11	-6	-5
2.5	18	1100	-13	-9	-10	-11	-8
2.8	18	1600	-14	-13	-10	-10	-16
2.5	17	1216	-12	-13	-15	-9	-14
2.4	17	1206	-11	-13	7	-9	-35
2.5	18	1262	-14	-17	-19	-16	-17
3.0	17	1130	-10	-12	-13	-9	-10
3.0	20	1006	-13	-14	-15	-16	-10
3.8	18	992	-11	-12	-9	-11	-13
3.8	21	1300	-15	-14	-18	-15	-14
3.6	19	1510	-12	-11	-10	-12	-12
4.2	20	1500	-9	-8	-8	-7	-11
3.5	21	1800	-14	-12	-15	-11	-10
2.6	18	1200	-9	-20	-14	-23	-34
3.2	18	1000	-7	-5	-5	-6	-5
6.4	20	1200	-7	-4	7	-7	-5
5.0	21	1106	-13	-16	-14	-7	-26

BASAL INFERIOR	MID INFERIOR	APICAL INFERIOR	APEX	3CHAMBER STRAIN	BASAL ANTEROSEPTAL	MID ANTEROSEPTAL	APICAL ANTEROSEPTAL	BASAL INFEROLATERAL
-10	-4	-7	-13	-8	5	-6	-12	-9
-14	-18	-15	-13	-10	-8	-14	-10	-8
-9	-7	-10	-13	-11	-9	-11	-11	-14
-8	-11	-9	-6	-9	-9	-7	-11	-9
-8	-13	-12	-11	-8	-11	-7	-8	-9
-11	-15	-14	-12	-14	-9	-17	-16	-12
-23	-37	-23	-35	-25	-20	-28	-33	-26
-15	-7	-8	-7	-18	-11	-5	-38	-5
-14	-18	-17	-10	-12	-10	-15	-12	-15
12	23	-20	-17	-9	9	12	-22	11
-6	9	-11	-8	-7	24	-27	-14	-21
-11	-9	-12	-7	-10	-11	-8	-15	-7
-6	-5	-6	-6	-8	-9	-9	-10	-7
-12	-10	-9	-11	-10	-13	-7	-9	-11
-14	-15	-10	-14	-9	-8	-12	-7	-10
-9	-14	-10	-11	-8	-9	-6	-8	-11
-15	-12	-13	-16	-11	-10	-13	-11	-9
-12	-9	-5	-8	-9	-9	-7	-7	-16
-8	-12	-14	-6	-13	-16	-12	-11	-9
12	-13	-2	-1	-8	-8	4	-13	-4
-9	-11	-12	-6	-11	-13	-11	-10	-13
-23	-4	-9	-13	-5	8	-13	-8	-8
-12	-12	-17	-17	-12	-12	-18	-18	-17
-17	-16	-10	-14	-11	-9	-10	-12	-13
-11	-13	-10	-6	-16	-21	-15	-14	-18
-14	-17	-13	-16	-14	-18	-12	-14	-15
-7	-8	-7	-5	-12	-14	-11	-13	-14
-12	-9	-10	-9	-8	-5	-24	-3	-6
-17	-12	-13	-14	-13	-17	-11	-10	-9
-15	-15	-8	-15	-12	-11	-9	-14	-13
-21	-9	-33	-34	-14	-14	-16	-22	-10
-20	-14	-15	-18	-12	-11	-8	-14	-15
-17	-14	-9	-12	-9	-11	-13	-7	-8
-14	-16	-15	-12	-15	-13	-11	-17	-18
-12	-15	-10	-6	-11	-9	-13	-11	-15
-13	-13	-15	-10	-15	-18	-17	-18	-13
-13	-11	-10	-9	-13	-13	-11	-10	-9
-9	-8	-7	-6	-10	-11	-12	-8	-7
-12	-11	-13	-12	-15	-17	-13	-15	-14
-21	-23	-9	-21	-4	8	-18	-11	6
-4	-7	-4	-4	-7	-8	-6	-10	-8
5	-9	-9	-7	-7	-5	8	-22	-19
-19	-5	-26	-26	-11	-10	-11	-19	-7

MID INFEROLATERAL	APICAL INFEROLATERAL	APEX	4CHAMBER STRAIN	BASAL ANTEROLATERAL	MID ANTEROLATERAL	APICAL ANTEROLATERAL	BASAL INFEROSEPTAL	MID INFEROSEPTAL
-5	-16	-14	-12	-10	-6	-21	-8	-4
-6	-12	-10	-9	-7	-8	-12	-14	-10
-10	-11	-11	-15	-16	-12	-14	-16	-18
-10	-8	-9	-8	-7	-8	-7	-9	-9
-8	-7	-6	-11	-8	-12	-10	-12	-13
-13	-18	-13	-15	-17	-10	-15	-18	-13
-25	-22	-27	-9	-11	-14	-9	-9	-11
-26	-21	-29	-11	-15	4	-16	-12	-7
-14	-10	-8	-18	-18	-21	-22	-16	-17
-16	-15	-17	-10	-6	-13	-18	-9	11
-13	6	-8	-11	-21	-6	-11	-4	-14
-10	-15	-14	-13	-13	-15	-11	-10	-16
-7	-8	-6	-9	-11	-7	-12	-9	-8
-12	-9	-10	-9	-7	-12	-9	-7	-10
-11	-9	-6	-15	-15	-20	-13	-11	-12
-7	-6	-9	-8	-7	-10	-6	-10	-8
-13	-12	-10	-14	-9	-18	-17	-15	-14
-10	-8	-6	-7	-7	-8	-12	-8	-6
-15	-13	-15	-7	-6	-8	-9	-7	-5
-10	-8	-11	-3	10	-18	-5	-16	-5
-12	-11	-7	-10	-9	-8	-13	-10	-9
-9	7	-5	-15	-16	-21	-14	-14	-24
-9	-9	-13	-16	-16	-13	-24	-17	-12
-8	-12	-13	-13	-10	-10	-15	-13	-12
-16	-10	-18	-13	-10	-9	-10	-14	-12
-17	-10	-12	-19	-21	-17	-16	-20	-23
-15	-10	-7	-11	-13	-9	-12	-11	-10
-9	-9	-6	-23	-18	-20	-36	-16	-15
-15	-16	-13	-16	-14	-17	-19	-11	-16
-12	-13	-12	-10	-13	-15	-7	-6	-10
5	-16	-14	-11	7	-12	-13	-10	-18
-13	-12	-11	-13	-17	-11	-10	-13	-15
-9	-15	-9	-9	-11	-7	-8	-9	-13
-15	-14	-17	-12	-15	-12	-10	-11	-14
-7	-10	-12	-10	-10	-9	-12	-8	-12
-19	-15	-15	-16	-11	-19	-17	-18	-16
-18	-13	-17	-12	-11	-13	-12	-9	-12
-13	-10	-9	-9	-9	-11	-8	-8	-12
-11	-17	-18	-15	-17	-13	-15	-14	-17
9	-15	-6	-6	-19	-22	-10	-8	-6
-7	-5	-5	-9	-10	-5	-15	-15	-5
6	-17	-20	-10	-10	-8	-10	-13	-7
-10	-13	-16	-12	-19	-6	-13	-18	-8

APICAL INFEROSEPTAL	APEX	POST CPB GCS	TG BASAL STRAIN	INFERIOR	INFEROSEPTAL	ANTEROSEPTAL	ANTERIOR	ANTERIOLATERAL
-16	-18	-18	-22	-24	-27	-20	-28	-13
-6	-6	-25	-25	-23	-28	-22	-32	-25
-17	-12	-20	-17	-18	-15	-16	-19	-18
-8	-8	-24	-20	-21	-23	-18	-21	-17
-9	-12	-23	-25	-24	-26	-31	-20	-25
-15	-17	-20	-21	-20	-16	-23	-21	-20
-9	-7	-30	-24	-9	-30	-19	-36	-17
-12	-14	-32	-32	-38	-32	-30	-30	-28
-23	-9	-25	-24	-20	-28	-25	-21	-26
-18	-16	-29	-26	-25	-18	-36	-23	-35
-18	-13	-25	-24	-24	-26	-28	-21	-23
-12	-14	-22	-18	-16	-15	-24	-18	-17
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-13	-13	-18	-17	-10	-18	-16	-23	-18
-13	-18	-26	-25	-40	-14	-21	-44	-13
-11	-9	-26	-24	-31	-27	-21	-22	-20
-12	-8	-19	-17	-22	-23	-18	-25	-15
-15	-21	-23	-20	-19	-15	-25	-22	-17
-9	-13	-23	-25	-27	-21	-23	-24	-20
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-17	-15	-27	-23	-21	-20	-36	-15	-20
-16	-9	-23	-22	-19	-23	-20	-17	-26
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-13	-14	-20	-19	-18	-19	-16	-20	-21
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-8	-9	-24	-17	-15	-27	-21	-25	-28
-3	-3	-18	-20	-20	-16	-22	-18	-24
-14	-12	-17	-20	-10	-18	-22	-34	-22
-19	-16	-25	-22	-22	-38	11	-31	-26

INFEROLATERAL	TG MID STRAIN	INFERIOR	INFEROSEPTAL	ANTEROSEPTAL	ANTERIOR	ANTEROLATERAL	INFEROLATERAL	TG APICAL STRAIN	INFERIOR	SEPTAL
-18	-12	-15	6	-17	-13	-15	-18	-20	-16	-16
-20	-30	-22	-27	-33	-28	-28	-28	-22	-24	-24
-16	-25	-28	-26	-25	-23	-27	-21	-18	-15	-17
-20	-28	-30	-25	-29	-23	-29	-32	-24	-28	-22
-24	-21	-20	-23	-21	-22	-18	-23	-23	-25	-21
-26	-17	-19	-20	-20	-26	-14	-13	-22	-24	-20
-46	-30	-33	-28	-34	-35	-36	-24	-39	-45	-35
-34	-29	-38	-27	-25	-45	-34	-26	-33	-34	-38
-24	-25	-26	-31	-23	-23	-17	-30	-26	-25	-23
-29	-28	-19	-26	-43	-30	-27	-31	-31	-30	-34
-22	-26	-28	-30	-22	-24	-26	-26	-25	-20	-28
-18	-26	-28	-24	-26	-21	-29	-28	-22	-19	-20
-24	-22	-25	-20	-19	-18	-28	-22	-21	-20	-19
-22	-23	-25	-21	-19	-23	-24	-26	-24	-26	-22
-24	-28	-30	-27	-28	-25	-31	-27	-23	-28	-21
-27	-23	-25	-26	-27	-21	-21	-19	-21	-20	-19
-17	-23	-20	-19	-27	-23	-22	-27	-19	-18	-17
-15	-22	-26	-20	-18	-19	-23	-26	-19	-20	-20
-20	-27	-30	-31	-29	-23	-23	-25	-23	-27	-20
-11	-19	-17	-18	-31	-18	-28	-11	-17	-24	-17
-18	-17	-15	-19	-20	-21	-17	-10	-17	-20	-21
-17	-17	-10	-18	-16	-23	-18	-17	-25	-27	-22
-35	-32	-34	-21	-30	-45	-35	-28	-20	-27	-14
-23	-26	-28	-25	-26	-27	-25	-24	-28	-20	-27
-20	-21	-17	-23	-21	-16	-24	-25	-16	-14	-19
-22	-24	-28	-25	-19	-26	-27	-19	-25	-20	-28
-20	-21	-19	-17	-25	-23	-21	-21	-23	-27	-23
-39	-32	-33	-35	-45	-35	-31	-27	-44	-48	-50
-20	-29	-32	-20	-27	-20	-19	-26	-22	-19	-26
-13	-22	-21	-19	-25	-26	-20	-21	-25	-31	-23
-29	-30	-33	-41	-39	-19	-22	-32	-22	-29	-32
-27	-25	-19	-27	-26	-24	-23	-31	-22	-24	-20
-22	-26	-27	-25	-27	-24	-25	-28	-25	-29	-21
-23	-19	-19	-23	-21	-18	-17	-16	-17	-19	-17
-23	-26	-27	-25	-26	-29	-27	-22	-27	-32	-27
-16	-22	-25	-23	-22	-21	-20	-21	-18	-21	-16
-20	-20	-19	-23	-20	-21	-18	-19	-21	-22	-20
-16	-26	-25	-28	-29	-26	-23	-25	-22	-25	-21
-19	-21	-19	-23	-21	-17	-25	-21	-16	-20	-15
8	-31	-31	-31	-36	-28	-41	-28	-25	-23	-33
-20	-16	-18	-10	-12	-18	-20	-16	-18	-18	-20
-13	-16	-17	-16	-17	-35	9	-17	-15	-15	-21
-24	-36	-41	-38	-32	-44	-25	-41	-17	-22	-30

ANTERIOR	LATERAL	POST OP	Vasopressor inotrope on ICU arrival	VIS on ICU arrival	DURATION OF VENTILATION (hrs)	ICU STAY (hrs)	PEAK VELOCITY (m/s)	MEAN GRADIENT (mm Hg)	POST OPERATIVE EF (%)
-22	-26		Noadr 0.05	5.0	12	44	1.80	18	58
-18	-22		No	0.0	16	42	1.50	10	50
-21	-19		Adr 0.02 Noradr 0.02	4.0	8	40	1.30	7	52
-22	-24		Adr 0.025 Noradr 0.025	5.0	17	45	1.70	11	80
-22	-24		No	0.0	11	43	2.50	17	75
-21	-22		Adr 0.03 Noradr 0.05	8.0	17	47	1.60	12	58
-49	-28		Adr 0.02 Noradr 0.05	7.0	12	62	2.30	6	60
-40	-29		Adr 0.05, Noradr 0.1	15.0	19	66	1.50	7	60
-28	-29		No	0.0	16	62	1.60	10	60
-27	-37		No	0.0	17	42	1.30	4	58
-26	-27		No	0.0	10	24	1.10	6	70
-22	-27		Adr 0.05, Noradr 0.05	10.0	10	26	1.20	8	57
-22	-23		Adr 0.1, Noradr 0.05	15.0	9	28	1.80	8	50
-27	-21		Adr 0.1, Noradr 0.05	15.0	14	28	2.30	7	68
-20	-23		Adr 0.02, Noradr 0.02	4.0	16	48	2.70	16	65
-22	-23		Adr 0.05, Noradr 0.01	6.0	15	28	2.60	13	56
-18	-19		Adr 0.05	5.0	18	29	2.40	11	69
-18	-18		No	0.0	23	43	2.60	11	58
-21	-24		No	0.0	16	26	1.50	8	56
-12	-20		No	0.0	11	40	1.30	10	71
-15	-12		No	0.0	10	24	1.90	17	50
-25	-26		Noradr 0.03	3.0	12	45	2.00	11	74
-21	-18		No	0.0	16	70	1.80	10	68
-26	-29		Noradr 0.02	2.0	10	78	2.80	15	67
-10	-21		No	0.0	5	48	2.20	11	62
-29	-23		No	0.0	12	68	1.40	5	69
-20	-22		Noradr 0.05	5.0	10	72	2.80	14	66
-45	-37		Noradr 0.05	5.0	14	20	1.20	9	65
-24	-19		No	0.0	16	42	2.00	11	58
-20	-26		Noradr 0.02	2.0	17	42	3.10	16	77
-25	-23		No	0.0	12	43	2.40	14	66
-26	-18		No	0.0	11	48	2.20	14	78
-25	-25		Noradr 0.05 Adr 0.02	7.0	15	48	2.10	11	59
-18	-14		No	0.0	14	40	2.83	19	55
-29	-20		Noradr 0.05	5.0	13	40	1.60	8	55
-18	-17		No	0.0	13	46	1.60	8	62
-19	-23		Noradr 0.02	2.0	16	48	2.90	20	60
-23	-19		No	0.0	11	48	1.60	6	68
-14	-15		Noradr 0.02	2.0	13	43	2.10	10	58
-29	18		Noradr 0.025 Adr 0.01	3.5	6	48	2.90	16	78
-22	-12		No	0.0	10	36	1.30	5	63
-15	-10		No	0.0	15	22	1.90	15	66
-8	-16		No	0.0	13	40	2.00	11	58

BASELINE DATA	PRE CPB	PRECPB GLS	2 CHAMBER STRAIN	3 CHAMBER STRAIN	4 CHAMBER STRAIN	PRE CPB GCS	TG BASAL
		-13	-18	-10	-9	-31	-31
		-9	-10	-8	-9	-31	-24
		-13	-15	-11	-12	-27	-24
		-7	-9	-8	-4	-21	-21
		-13	-15	-12	-12	-27	-23
		-12	-14	-10	-12	-28	-26
		-15	-18	-12	-15	-19	-19
		-15	-18	-12	-15	-24	-22
		-13	-15	-13	-17	-21	-20
		-14	-17	-11	-14	-20	-20

TG MID	TG APICAL	POST CPB	POST CPB GLS	2 CHAMBER STRAIN	3 CHAMBER STRAIN	4 CHAMBER STRAIN	POST CPB GCS	TG BASAL	TG MID	TG APICAL
-28	-35		-10	-9	-8	-12	-18	-22	-12	-20
-38	-30		-8	-8	-9	-8	-24	-20	-28	-24
-26	-30		-20	-28	-25	-9	-30	-24	-30	-39
-19	-23		-8	-6	-9	-10	-29	-26	-28	-31
-28	-30		-12	-12	-9	-15	-25	-24	-28	-23
-30	-28		-13	-15	-11	-13	-26	-24	-26	-28
-26	-27		-16	-15	-14	-19	-23	-20	-24	-25
-26	-24		-14	-13	-13	-16	-22	-20	-29	-22
-22	-21		-13	-14	-15	-12	-19	-21	-19	-17
-23	-20		-14	-12	-15	-15	-19	-20	-21	-16

INTRA OBSERVER VARIABILITY DATA	PRE CPB	PRECPB GLS	2 CHAMBER STRAIN	3 CHAMBER STRAIN	4 CHAMBER STRAIN	PRE CPB GCS	TG BASAL
		-12	-17	-9	-10	-32	-31
		-10	-10	-11	-9	-30	-26
		-14	-15	-12	-15	-26	-25
		-8	-10	-8	-6	-21	-19
		-12	-12	-13	-12	-28	-25
		-12	-13	-11	-12	-28	-28
		-16	-18	-14	-16	-20	-20
		-14	-16	-14	-12	-24	-23
		-13	-13	-15	-11	-20	-20
		-15	-18	-15	-12	-22	-22

TG MID	TG APICAL	POST CPB	POST CPB GLS	2 CHAMBER STRAIN	3 CHAMBER STRAIN	4 CHAMBER STRAIN	POST CPB GCS	TG BASAL	TG MID	TG APICAL
-32	-33		-11	-10	-12	-11	-16	-19	-14	-15
-34	-30		-8	-10	-7	-7	-25	-22	-29	-24
-27	-26		-20	-24	-22	-14	-28	-28	-30	-27
-21	-23		-8	-8	-7	-9	-30	-27	-31	-32
-29	-30		-12	-11	-10	-15	-26	-27	-25	-27
-30	-26		-12	-14	-11	-11	-25	-25	-26	-24
-19	-21		-17	-16	-15	-20	-23	-21	-23	-25
-25	-24		-14	-14	-14	-15	-23	-21	-27	-21
-21	-19		-15	-14	-16	-15	-19	-20	-21	-19
-24	-20		-14	-15	-12	-15	-18	-19	-20	-16

INTEROBSERVER VARIABILITY DATA	PRE CPB	PRECPB GLS	2 CHAMBER STRAIN	3 CHAMBER STRAIN	4 CHAMBER STRAIN	PRE CPB GCS	TG BASAL	TG MID	TG APICAL
		-13	-18	-11	-9	-32	-31	-30	-35
		-10	-10	-9	-11	-32	-26	-38	-32
		-14	-15	-12	-15	-27	-24	-26	-30
		-7	-7	-8	-7	-20	-20	-19	-21
		-13	-16	-11	-12	-26	-24	-27	-27
		-11	-13	-11	-10	-29	-27	-30	-30
		-15	17	-13	-15	-20	-21	-19	-20
		-14	-17	-12	-13	-22	-23	-20	-22
		-12	-13	-13	-10	-21	-20	-21	-22
		-15	-15	-16	-14	-21	-21	-22	-20

POST CPB	POST CPB GLS	2 CHAMBER STRAIN	3 CHAMBER STRAIN	4 CHAMBER STRAIN	POST CPB GCS	TG BASAL	TG MID	TG APICAL
	-9	-9	-8	-10	-20	-22	-16	-22
	-8	-9	-8	-8	-25	-23	-27	-25
	-19	-26	-23	-8	-31	-26	-31	-36
	-8	-7	-9	-8	-30	-26	-30	-33
	-12	-12	-10	-14	-26	-26	-28	-24
	-13	-14	-12	-13	-25	-25	-27	-23
	-16	-14	-15	-19	-24	-21	-25	-26
	-14	-14	-13	-15	-23	-23	-25	-20
	-13	-15	-14	-10	-19	-19	-21	-17
	-14	-13	-15	-14	-20	-22	-18	-19

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