Original Article

Comparison of Effects of Sevoflurane Versus Propofol on Left Ventricular Longitudinal Global and Regional Strain in Patients Undergoing On-Pump Coronary Artery Bypass Grafting

Chennakeshavallu G N, Shrinivas Gadhinglajkar, Rupa Sreedhar, Saravana Babu, Sruthi Sankar, Prasanta Kumar Dash

Department of Cardiac Anesthesia, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala, India

ABSTRACT

Background: Assessment of myocardial deformation by quantifying peak systolic longitudinal strain (PSLS) is a sensitive and robust index to detect subclinical myocardial dysfunction. We hypothesize that sevoflurane by virtue of anesthetic preconditioning preserves myocardial function better than propofol.

Aims: The authors have assessed the effects of sevoflurane and propofol on global longitudinal strain (GLS) as a primary outcome in patients undergoing on-pump coronary artery bypass grafting. Our secondary aim was to assess the pattern of regional distribution of segmental PSLS between the groups.

Materials and Methods: Fifty patients with normal left ventricular function undergoing coronary artery bypass grafting were analyzed in this prospective observational study. Consecutive patients received either propofol (P) or sevoflurane (S) anesthesia.

Measurements: Trans-esophageal echocardiographic images (mid-esophageal four-chamber, two-chamber, and three-chamber (long-axis)) were recorded during the precardiopulmonary bypass (CPB) and post-CPB period. Strain analysis (GLS/segmental PSLS) was done offline by investigators blinded to the study. The inotropic score, duration of inotropic support, and mechanical ventilation required were recorded.

Results: Following cardiopulmonary bypass and coronary revascularization, GLS reduced significantly in both the groups ($P < 0.05$). In the S-group, significant reduction in segmental strain was observed only in apical segments including apex, whereas in P-group significant reduction in segmental strain was seen in mid- and apical segments. The postoperative VIS, duration of inotropes/vasopressor required, and mechanical ventilation were similar in both the groups.

Conclusions: There are no significant differences in global left ventricular function as assessed by GLS between patients anesthetized with sevoflurane or propofol. However, regional PSLS was better preserved in the S-group compared to P-group.

Keywords: Coronary artery bypass grafting, propofol, sevoflurane, strain imaging, transesophageal echocardiography

Address for correspondence: Dr. Shrinivas Gadhinglajkar, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum - 695 011, Kerala, India.
E-mail: drgadhinglajkar@gmail.com

Submitted: 19-Sep-2020 **Revised:** 19-Sep-2020 **Accepted:** 06-Feb-2021 **Published:** 11-Apr-2022

Access this article online	
Quick Response Code:	Website: www.annals.in
	DOI: 10.4103/aca.aca_240_20

How to cite this article: Chennakeshavallu GN, Gadhinglajkar S, Sreedhar R, Babu S, Sankar S, Dash PK. Comparison of effects of sevoflurane versus propofol on left ventricular longitudinal global and regional strain in patients undergoing on-pump coronary artery bypass grafting. Ann Card Anaesth 2022;25:188-95.

INTRODUCTION

Reduction in left ventricular (LV) wall motion and global function after coronary artery bypass grafting (CABG) are associated with increased morbidity and mortality.[1-3] CABG performed using cardiopulmonary bypass (CPB), also called on-pump CABG, may be associated with impairment in the LV function, which may be attributed to the cardioplegia-induced myocardial electromechanical quiescence and ischemia-reperfusion injury.[4,5] Recent evidence suggests that sevoflurane exert protective effects on myocardium during ischemia and reperfusion.[6] Ischemia-induced metabolic changes in the myocardium are attenuated with administration of sevoflurane, which leads to improvement in the systemic hemodynamic parameters.[7] Although a number of studies have suggested that propofol may exhibit free radical scavenging properties,[8] the cardioprotective effects of propofol have not been well established.[9,10] We hypothesized that sevoflurane by virtue of its cardioprotective effects can attenuate myocardial damage better than propofol.

Echocardiographic assessment of LV systolic function is routinely performed using parameters such as two-dimensional (2D) LV ejection fraction (EF) and wall motion severity index (WMSI). The calculation of LVEF is based on the changes in the LV cavity volume. It does not consider the function of longitudinal myocardial fibers, which is affected in early stages of ischemia.[11] Strain analysis performed using 2D transesophageal echocardiography (TEE) enables a semiobjective and quantitative assessment of regional myocardial function, which is less preload-dependent than the LVEF.[12] Global longitudinal strain (GLS) detects subtle changes in myocardial dysfunction by quantifying the longitudinal myocardial deformation and has been shown to be more reproducible and more useful clinically than circumferential and radial strain.[13,14] GLS also has been demonstrated to be as accurate as sono-micrometry and magnetic resonance imaging in several conditions.[15] Segmental peak systolic longitudinal strain (PSLS) in 17-segment model reflects the reduction in regional ventricular wall motion better than the WMSI. Applications of Speckle-tracking strain imaging in the peri-operative period of cardiac surgery are still under evaluation. We conducted a prospective study to assess changes in the LV strain before and after CPB with two anesthesia techniques in patients undergoing CABG. The primary aim of this study was to compare the effect of sevoflurane and propofol on GLS in patients undergoing on-pump CABG. The secondary aim was to study the change in regional myocardial function induced by these anesthetic agents on segmental PSLS.

MATERIAL AND METHODS

The study was performed in adult patients scheduled for elective CABG, after obtaining institutional ethics committee approval and informed consent from the patients. The study was registered with Clinical Trial Registry of India (CTRI/2017/12/010823). Inclusion criteria were patients with preoperative LV EF >50% undergoing isolated elective CABG. Patients with left main disease >70%, poor LV function (EF <35%), arrhythmias, and concomitant valve diseases were excluded from the study. Consecutive patients received either propofol (P) or sevoflurane (S) anesthesia.

Patients were advised to continue all medications except oral hypoglycemic drugs, clopidrogel, and angiotensin-converting enzyme inhibitors. Oral diazepam 5–10 mg was administered for sedation the night before surgery. Patients in P-group received TIVA throughout the surgery. Propofol 1–2 mg/kg, fentanyl 5–10 μg/kg, midazolam 0.1 mg/kg, and pancuronium 0.1 mg/kg were used for anesthesia induction in P-group. General anesthesia was maintained with propofol infusion 75–100 μg/kg/min in a dedicated intravenous line throughout the procedure including during CPB. Bispectral index (BIS) values were maintained in the range of 40–50 throughout the surgery. No volatile anesthetic was administered at any time during the procedure. The induction of general anesthesia in the S-group consisted of fentanyl 5–10 μg/kg, midazolam 0.1 mg/kg, pancuronium 0.1 mg/kg, and etomidate 0.2 mg/kg. Anesthesia was maintained with sevoflurane at an end-tidal concentration of at least one minimum alveolar concentration throughout the procedure, including during CPB, and was guided by the BIS value, which was maintained between the range of 40 and 50. During CPB, sevoflurane was administered in the oxygenator circuit through a dedicated calibrated vaporizer. In pre-CPB period, any hypertension or hypotension was treated with nitroglycerin infusion or phenylephrine boluses, respectively, to maintain the target mean arterial pressure (MAP) ≥65 mmHg. All patients were mechanically ventilated with volume control ventilation mode. Intraoperative continuous monitoring in both the groups included five-lead electrocardiography with ST-segment analysis, heart rate, invasive blood pressure, pulse oximetry, core and nasopharyngeal temperatures, respiratory gas monitoring, central venous pressure, and BIS. All patients underwent on-pump CABG using JostraHL20 (Maquet, Rastatt, Germany) heart lung machines with a roller pump for non-pulsatile perfusion. Electromechanical quiescence was achieved using intermittent antegrade cold blood cardioplegia. Two surgeons were involved in the study period as the first

operators. At CPB weaning, as per institutional protocol, adrenaline infusion was commenced at a starting dose of 0.05 μg/kg/min if MAP was less than 65 mm Hg, and TEE-guided cardiac index (CI) at LV outflow tract was less than 2 L/min/m^2. The decision to further add the inotropes and vasopressor was left to the discretion of anesthesiologist who was not the part of the study. At the end of the surgical procedure, patients were transferred to the intensive care unit and kept sedated with a morphine infusion of 20 μg/kg/h. When hemodynamically stable and rewarmed, the patients were weaned from the ventilator and extubated. All TEE examinations were performed before sternotomy (pre-CPB) and at the time of skin closure (post-CPB) with three-dimensional matrix array probe coupled with an ultrasound system (IE 33, Philips Healthcare, Bothell, WA). Echocardiographic measurements were performed by a cardiac anesthesiologist who was blinded to the patient's clinical information.

Peak GLS and segmental strain were estimated using video clips recorded at transthoracic apical 4-chamber view, apical two-chamber view, and long-axis view to obtain baseline values. Video clips were also recorded at ME four-chamber view, ME two-chamber view, and ME three-chamber (LAX) view to obtain pre-CPB and post-CPB values with similar heart rates (within 10 beats/min) as described by EACVI/ASE.[16,17] A single-lead electrocardiogram (lead I/II) was recorded simultaneously. Imaging depth and sector width were adjusted to achieve a frame rate >50 frames/s. Images displaying clear endocardial and epicardial borders and minimum artifacts were selected for processing. Three cardiac cycles were analyzed for each imaging plane and stored as cine-loops in DICOM format. For each measured or calculated variable, the averages of the three measurements were reported. Strain analysis was performed offline using a dedicated software package (QLAB 9, Philips Medical Systems—Andover, MA, USA) with the semiautomatic delineation of endocardial and epicardial borders, which tracked the speckles throughout the cardiac cycle and derived the GLS. A three-click method was used for anchoring three points in ME four-chamber view and ME two-chamber view, wherein two points were placed on both sides of the mitral annulus and third at the apex of the LV. This simplified the process of tracking and analyzing peak systolic strain. For ME-LAX view, the points were placed at posterior mitral annulus, aortic annulus near right coronary cusp, and LV apex. A color-coded parametric image was generated by the software that provided quick visual impression of the extent of segmental myocardial deformation. The timing of aortic valve closure was automatically determined. The quality of the tracking was visually assessed by the operator during motion playback. If necessary, the width of the region of interest was manually adjusted to encompass the myocardial thickness excluding pericardium. Segments with inadequate tracking were rejected. The processed data were displayed in bull's eye format [Figure 1]. All published literature on strain quantification accords a negative value to the longitudinal strain. An increase in longitudinal strain is denoted by the value becoming more negative and a decrease in the strain by the value becoming less negative.

The decision regarding extubation and duration of inotropes required was left at the discretion of attending intensivist, who was not the part of the study. The vasoactive inotropic score (VIS) was determined on arrival to ICU using the standard formula.[18]

Vasopressor inotropic score = Dopamine dose (μg/kg/min) + Dobutamine dose (μg/kg/min) + 100 × Adrenaline (μg/kg/min) + 100 × Norepinephrine (μg/kg/min) + 10,000 × Vasopressin (μg/kg/min).

Statistical analysis

A minimum difference of − 1.5% of GLS between the pre- and post-CPB period in a pilot study was observed. Sample-size calculation was based on a two-sided alpha error of 0.05 and power of 80%. To detect a difference in LV GLS of − 1.5% units between the pre-CPB and post-CPB period, 18 patients were needed to be included in each group at a standard deviation of mean difference of paired measurement of two. Considering a dropout rate of 20%, we included 23 patients in each group. The results obtained from the study were expressed in tabular format.

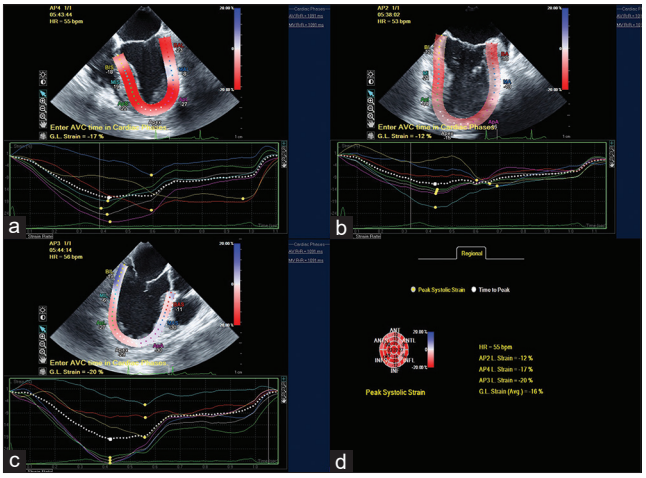

Figure 1: Representative 2D longitudinal strain imaging depicting GLS and segmental PSLS. The time–longitudinal strain curves for the ME-4ch (a), 2-ch (b), and 3-ch (c) views are displayed. In (d), the bull's eye plot displays the regional value of PSLS for 17 segments and the LV GLS is averaged. Abbreviations: PSLS: peak systolic longitudinal strain, GLS: Global longitudinal strain.

Our data were normally distributed and expressed as mean ± SD. Quantitative variables such as segmental PSLS, GLS, and inotropic score were expressed as mean ± SD. Pre-CPB and post-CPB comparison of quantitative variables were analyzed by paired *t* test or Wilcoxon sign-rank test. Intergroup comparisons of quantitative variables were compared with an unpaired *t* test and Fisher's exact test where appropriate. A *P* value of <0.05 was considered as statistically significant. Statistical analysis was performed using SPSS version 22.0.

RESULTS

In this prospective observational study, a total of 71 patients were included. Twenty-one patients were excluded for the following reasons: patient not willing to participate ($n = 5$), poor quality images, which were difficult to analyze in post-bypass period ($n = 12$), and frequent ventricular premature complexes/irregular cardiac cycle (atrial fibrillation) that precluded strain imaging in post-bypass period ($n = 3$). One patient required additional graft placement under second run of CPB, who also was excluded from the study. There were no differences in demographic details, comorbidities, and frequency of coronary artery diseases between the groups. The baseline TTE echocardiographic parameters (EF, WMSI, CI, and lateral S' velocity, and GLS) were comparable between the groups. Intraoperatively, the number of venous grafts and arterial grafts placed were statistically similar between the groups. The mean aortic cross-clamp and CPB time were similar [Table 1].

Changes in segmental PSLS and GLS in both the groups before and after CPB are presented in Table 2. GLS decreased significantly after CPB and revascularization in both the groups ($P < 0.05$) compared to pre-CPB values. In S-group, GLS decreased from − 13.38% ± 3.72 to − 11.69% ± 2.76 ($P = 0.0051$), whereas in P-group, GLS decreased from − 14.75% ± 3.24 to − 11.93% ± 3.11 ($P < 0.0001$). In both the S- and P-groups, there was a statistically insignificant decrease in PSLS in the basal segments ($P > 0.05$). In mid-segments, the patients in S-group had insignificant decrease in PSLS ($P = 0.5816$). Mid segmental PSLS in the P-group decreased significantly from −14.25% ± 2.81 to − 12.90% ± 2.59 ($P = 0.0285$). The apical segments PSLS including the apex showed decrease significantly in post-CPB ($P < 0.05$) in both the groups. There was a decrease in apical segments PSLS by −2.86% and −4.23% units in S- and P-group, respectively.

PSLS of individual LV myocardial segments with mean difference from pre- to post-CPB values in both the groups is displayed in Figure 2 in standard bull's eye format of LV segmental anatomy (17-segment model). When the mean differences between the pre-CPB and post-CPB values in individual anatomical segments were averaged in S-group, only the apical segments and the apex showed a large PSLS reduction in post-CPB period ($P < 0.05$), whereas the mid and basal segments showed statistically insignificant reduction. However, in the P-group, significant reductions in PSLS were noted in the mid and apical segments

Table 1: Patient characteristics

Parameter	S-Group (*n*=26) Mean±SD *n* (%)	P-group (*n*=24) Mean±SD *n* (%)	*P* (*P*<0.05 significant)
Preoperative data			
Age (years)	59.27±9.84	61.46±9.01	0.417
Sex, F/M	7/19	5/19	0.628
Body mass index (kg/m^2)	22.4±4.2	20.3±4.3	0.15
Diabetes	20 (77%)	18 (75%)	0.869
Hypertension	14 (53%)	19 (79%)	0.055
NYHA I/II/III/IV	0/19/7/0	0/19/5/0	0.6187
Coronary angiogram			
LMCA>70% stenosis	0	0	
LAD>70% stenosis	25	24	0.336
LCX>70% stenosis	18	20	0.253
RCA>70% stenosis	24	20	0.338
Intraoperative data			
No of venous bypass grafts (median)	3 (2-4)	3 (2-4)	1
No. of arterial grafts (median)	1	1	1
Aortic cross clamp (min)	45.76±5.399	46.53±6.28	0.643
CPB time (min)	107.56±13.87	111.65±12.86	0.286
Preoperative transthoracic echocardiogram data			
Ejection fraction (%)	53.1±4.70	54.7±5.10	0.7741
Wall motion score	1.21±0.22	1.15±0.23	0.3506
Cardiac index (L/m^2)	2.8±0.64	2.8±0.58	1
Lateral S' velocity (m/s)	6.09±1.41	6.59±1.05	0.1642
GLS	−16.02±4.90	−15.45±5.03	0.6867

Data presented as mean±SD, number/percentage and median (range). Unpaired *t* test and Fisher's exact test were applied

including the apex ($P < 0.05$). Intergroup comparison of GLS and segmental strain were similar in both pre-CPB and post-CPB period [Table 3]. There were no significant differences between the groups with respect to VIS, duration of inotropes required, and mechanical ventilation in ICU [Table 4]. Twenty patients were analyzed for intraobserver and interobserver variability. Intraobserver interclass coefficients (ICCs) with 95% confidence intervals indicated good reliability in pre-CPB strain and moderate reliability in post-CPB strain measurements. Interobserver ICCs were lower than intraobserver ICCs in both pre- and post-CPB in all the segments with moderately good agreement [Table 5].

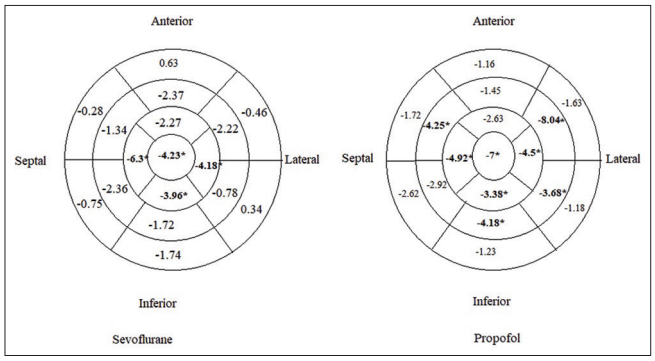

Figure 2: Left ventricular segmental changes in myocardial longitudinal strain (17-segment model) in S- and P-group. The mean difference in segmental longitudinal strain values between pre- and post-CPB period is displayed in each segment. *Represents statistically significant decrease in longitudinal strain from pre- to post-CPB period ($P < 0.05$).

DISCUSSION

In this prospective study, we compared the effects of sevoflurane and propofol at equi-anesthetic doses (BIS = 40–50) on segmental PSLS and GLS in patients undergoing CABG. Patients received either

Table 2: Pre- and post-CPB comparison of GLS and segmental PSLS in both the groups

Strain	Sevoflurane (*n*=26)			Propofol (*n*=24)		
	Pre-CPB (mean, SD)	Post-CPB (mean, SD)	*P*	Pre-CPB (mean, SD)	Post-CPB (mean, SD)	*P*
BA	−11.53, 4.55	−12.16, 4.67	0.6390	−12.33, 3.85	−11.17, 3.58	0.2970
BAS	−10.96, 4.24	−10.68, 3.81	0.8124	−11, 4.38	−9.28, 3.43	0.0937
BIS	−12.53, 3.78	−11.78, 5.37	0.5682	−12.70, 4.01	−10.08, 5.03	0.0562
BI	−14.30, 4.97	−12.56, 6.17	0.2759	−11.83, 5.41	−10.60, 4.69	0.4164
BIL	−13.38, 4.88	−13.72, 5.31	0.8110	−14.08, 5.28	−12.90, 4.58	0.4244
BAL	−14.03, 4.76	−14.27, 6.55	0.8821	−14, 4.31	−12.37, 6.35	0.3104
MA	−14.73, 4.78	−12.36, 5.38	0.1056	−15.54, 6.37	−14.09, 8.62	0.5174
MAS	−15.50, 5.51	−14.16, 7.77	0.4825	−15.16, 4.99	−10.91, 4.35	0.0037*
MIS	−15.53, 3.84	−13.17, 5.44	0.0768	−13.62, 4.98	−10.70, 5.66	0.0666
MI	−16.29, 5.11	−14.57, 4.36	0.1977	−15.54, 6.57	−11.36, 3.68	0.0022*
MIL	−14.73, 4.92	−13.95, 7.03	0.1096	−16.37, 6.92	−12.69, 4.86	0.0412*
MAL	−14.69, 5.19	−12.47, 5.80	0.1070	−19.79, 6.75	−11.75, 4.55	<0.0001*
AA	−16.19, 4.26	−13.92, 4.47	0.0722	−17, 5.64	−14.37, 6.21	0.1352
AS	−17.69, 5.02	−11.39, 3.71	0.0005*	−19.25, 7.15	−14.33, 6.47	0.0174*
AI	−17.92, 4.90	−13.96, 4.85	0.0061*	−16.83, 4.89	−13.45, 5.15	0.0025*
AL	−19.34, 5.16	−15.16, 7.10	0.0206*	−18.54, 4.86	−14.04, 5.96	0.0067*
Apex	−17.84, 5.94	−13.61, 5.76	0.0012*	−18.54, 5.74	−11.54, 5.00	0.0001*
GLS	−13.38, 3.72	−11.46, 2.74	0.0051*	−14.75, 3.24	−11.93, 3.11	<0.0001*

* represents statistically significant decrease in regional PSLS ($P < 0.05$). Data expressed as mean±SD. Paired *t*-test was applied to compare pre- and post-CPB values. Abbreviations: BA: basal anterior, BAS: basal anterioseptal, BIS: basal inferoseptal, BI: basal inferior, BAL: basal anterolateral, MA: mid anterior, MAS: mid anteroseptal, MIS: mid inferoseptal, MI: mid inferior, MIL: mid inferolateral, MAL: mid anterolateral, AA: apical anterior, AS: apical septal, AI: apical inferior, AL: apical anterior, GLS: global longitudinal strain, PSLS: peak systolic longitudinal strain

Table 3: Intergroup comparison of GLS and segmental PSLS between the groups

	Pre -CPB			Post-CPB		
	Sevoflurane	Propofol	*P*	Sevoflurane	Propofol	*P*
GLS	−13.38% ± 3.72	−14.75% ± 3.24	0.2155	−11.69% ± 2.76	−11.93% ± 3.11	0.4874
Basal segments PSLS	−12.03±3.15	−13.37±2.33	0.1473	−11.73±2.60	−12.14% ± 2.18	0.7731
Mid segments PSLS	−13.15±3.28	−14.25±2.81	0.3653	−12.19±3.25	−12.90±2.59	0.6593
Apical segments PSLS	−17.30±3.08	−18.29±3.329	0.4071	−14.44±3.39	−14.13±4.32	0.3638

Data expressed as mean±SD. Unpaired t-test was applied

Table 4: Postoperative parameters between the groups

Parameter	Sevoflurane (*n*=26)	Propofol (*n*=24)	*P*
Vasoactive inotropic score	5.19±3.15	5.52±3.53	0.5242
Mechanical ventilation duration (h)	5.17±1.82	6.12±2.15	0.6283
Duration of inotropic support	24.19±14.20	28.39±13.29	0.2099

Data presented as mean±SD. Unpaired *t*-test was applied

Table 5: Intraobserver and interobserver variability of GLS and segmental PSLS

Longitudinal strain	Pre-CPB strain (95% CI)	Post-CPB strain (95% CI)
Intraobserver		
GLS	0.78 (0.64-0.80)	0.74 (0.60-0.76)
Basal segments PSLS	0.78 (0.67-0.75)	0.73 (0.61-0.78)
Mid segments PSLS	0.79 (0.68-0.81)	0.73 (0.64-0.79)
Apical segments PSLS (apex)	0.81 (0.70-0.83)	0.75 (0.72-0.80)
Interobserver		
GLS	0.65 (0.55-0.73)	0.60 (0.53-0.73)
Basal segments PSLS	0.55 (0.42-0.69)	0.52 (0.44-0.66)
Mid segments PSLS	0.69 (0.55-0.80)	0.66 (0.53-0.76)
Apical segments PSLS (apex)	0.70 (0.58-0.78)	0.65 (0.55-0.74)

Data presented as intraclass correlation coefficient (95% CIs) for global and segmental longitudinal strain at pre- and post-CPB period

sevoflurane or propofol throughout the intraoperative period titrated to BIS value of 40–50. Other factors, which affect the myocardial function like fentanyl dosage, CPB duration, aortic cross-clamp time, and number of coronary bypass grafts, were constant during the study period. Since strain is affected by loading condition, effects of these agents on myocardial strain were assessed at constant loading conditions, maintaining baseline hemodynamic values in both groups within ±20% of the limits. Segmental PSLS and GLS were compared between the groups during pre-CPB and post-CPB.

Many factors are known to influence the perioperative LV systolic function during CABG surgery. These include patient characteristics such as age, extent of coronary artery disease, and degree of underlying LV dysfunction and surgery-related events such as number and quality of grafts, type of cardioprotection, duration of aortic cross-clamp, and CPB. In our study, patient characteristics and surgical features were similar in both the groups. This suggests that the differences in cardiac function between the groups were not associated with patient's characteristics and intraoperative events but with the choice of anesthetic agents. Even though opioids have been shown to offer cardioprotective effects similar to that of ischemic preconditioning,[19] we do not attribute the observed differences in cardiac function between the groups to fentanyl as it was administered in similar dosage in both the groups. Various studies have reported utility of conventional echocardiographic techniques for the evaluation of effects of sevoflurane and propofol on LV global and regional systolic function. The commonly employed intraoperative method of WMSI that quantifies regional myocardial function has many limitations: (1) it is highly subjective, operator-dependent, and scoring is based upon qualitative evaluation of wall motion. (2) It focuses only on the function of radial myocardial fibers without taking into account the function of longitudinal fibers, which are involved first at early stages of myocardial ischemia and dysfunction. Even LVEF, a frequently used quantitative parameter of LV systolic function, is deranged only when a significant number of myocardial segments become dysfunctional. Hence, conventional methods of echocardiography do not provide insight into the contractile dysfunction of different categories of myocardial fibers.

As a novel technique, speckle-tracking imaging has been used recently to evaluate global and regional myocardial function, and it can provide reproducible data on myocardial deformation, not only in radial and circumferential directions but also in the longitudinal direction.[20,21] Longitudinal strain on speckle-tracking imaging can quantitatively measure the elongation force of the subendocardial myocardium in the longitudinal direction.[22] Longitudinal strain can assess global and regional LV systolic function with good accuracy and reproducibility.[23] Therefore, strain quantification may be regarded as a reference method for the assessment of regional myocardial function.

In the present study, we observed reduction in GLS after anesthesia induction in both the groups compared to awake GLS values. The probable cause of reduction in GLS was twofold: first, changes in myocardial loading condition, myocardial depression induced by anesthetic agents and initiation of mechanical ventilation, and other reason being differences in GLS as they were measured using TTE and TTE modalities. Dalla *et al.*[24] demonstrated that reduction occurs in LV GLS after anesthesia induction and initiation of positive pressure ventilation. Badran *et al.*[25] compared TTE and TEE echo modalities in evaluation of LV deformation and concluded that regional and GLS measured by TTE showed higher values compared with its corresponding values measured by TEE.

We noticed an overall significant decrease in GLS after CPB in both the groups. After CPB and revascularization, the GLS was reduced in both the groups to a similar extent. The reduction in post-CPB GLS can be attributed to residual effects of cardioplegia, myocardial stunning,

episodes of subendocardial ischemia during CPB, and ischemia reperfusion injury. Subendocardial myocardial ischemia is the leading cause of decreased longitudinal strain.[26] In patients undergoing CABG, variable patterns of recovery of ventricular function have been reported in the postoperative period. Most studies describe a decrease in ventricular function between 2 and 6 h after operation, with a return to normal within 24 h to 7 days.[27,28] No difference was observed in the pre-CPB GLS between the groups in our patients. In immediate post-CPB period, the GLS was reduced by −1.69% units in S-group and by −2.82% units in P-group when compared to pre-CPB values which was statistically insignificant ($P = 0.4874$). Although better preservation of myocardial function was reported in sevoflurane group than the propofol after CABG,[29] in some of the published studies, we did not observe this difference as assessed with GLS. Propofol and sevoflurane might provide protection to the adult myocardium by different mechanisms. Propofol decreases postischemic myocardial mechanical dysfunction, infarct size, and histological degeneration. It also suppresses the activity of neutrophils and may therefore produce its beneficial effects by reducing free radicals, Ca^{2+} influx, and neutrophil activity.[30] On the other hand, sevoflurane improves recovery of contractile function of the stunned myocardium by a mechanism similar to ischemic-preconditioning, with an improvement in the postischemic contractility.[31]

The magnitude and pattern of segmental PSLS are heterogeneous in patients with coronary artery disease. The values for segmental PSLS did not remain constant in all myocardial segments but increased gradually from base to apex. Sutherland *et al*.[32] mentioned that the longitudinal peak systolic strain is often (but not always) marginally higher in the mid segments than in the basal segments in all four cardiac walls. In both of our groups, we found an increased gradient (from pre- to post-CPB) in segmental PSLS from the LV base to apex, with smallest decrements in the ventricular base and largest in the apex. This is attributed to decreased coronary perfusion in distal vascular beds during cardioplegia administration, which could have led to increased subendocardial ischemia. As coronary artery perfusion pressure is related to vessel lumen area and systemic perfusion pressure, it is likely that perfusion of vascular bed in the distal apical areas was more compromised than in the basal segmental areas during CPB. We do not attribute the differential pattern of PSLS in our subjects to normal heterogeneity in regional myocardial strain, as we observed the mean differences in PSLS values between the post-CPB period and pre-CPB period. After CPB and revascularization in S-group, the segmental PSLS detected reduction in myocardial function only in the apical segments and apex. However, in P-group, there was reduction in segmental PSLS in mid and apical segments including apex after CPB. This can be partly explained by variation in patterns of coronary artery lesions and degree of collateralization, which was not addressed in this study or can be attributed to better myocardial protective effects of sevoflurane during episodes of ischemia and reperfusion during CPB.

In both groups, all patients needed inotropic and vasoconstrictive support after CPB and in the first hours in the intensive care unit. The VIS, duration of inotropes/vasopressor requirement, and extubation time did not differ between the groups. Similar results were reported in a meta-analysis conducted by Yao *et al*.[33]

We do acknowledge limitations to our study. Given the small sample size of our cohort, further large studies are required to validate these findings. Due to technological limitations, the strain quantification was performed offline in our study, unlike the routine parameters such as LVEF, WMSI, and CI that could be derived in the intraoperative period. The technology of strain quantification remains vendor-specific and there is no consensus among the vendors on strain analysis. We did not measure the cardiac biomarkers, which could have reinforced our observations pertaining to the changes in longitudinal strain that occurred with two different anesthetic regimens, since it is not a part of our institutional practice to quantify cardiac biomarkers in postoperative period routinely. Also, we did not follow-up the patients to ascertain whether the regional LV function would be better preserved in sevoflurane group, which may have impact on long-term postoperative function.

In summary, our study suggests that there is no difference between sevoflurane and propofol at BIS-guided anesthetic doses on GLS in low-risk patients undergoing CABG and having good baseline LV function. However, the segmental PSLS was better preserved in sevoflurane group. Further large randomized studies are needed to evaluate the effect of increasing dose of sevoflurane and propofol on GLS and segmental PSLS in patients undergoing CABG including those with reduced LV function.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Ascione R, Narayan P, Rogers CA, Lim KH, Capoun R, Angelini GD.

Early and midterm clinical outcome in patients with severe left ventricular dysfunction undergoing coronary artery surgery. Ann Thorac Surg 2003;76:793-9.
2. Roques F, Nashef SA, Michel P, Gauducheau E, de Vincentiis C, Baudet E, *et al*. Risk factors and outcome in European cardiac surgery: Analysis of the Euro SCORE multinational database of 19030 patients. Eur J Cardiothorac Surg 1999;15:816–23.
3. Hamad MA, Van Straten AH, Schönberger JP, ter Woorst JF, de Wolf AM, Martens EJ, *et al*. Preoperative ejection fraction as a predictor of survival after coronary artery bypass grafting: Comparison with a matched general population. J Cardiothorac Surg 2010;5:29.
4. Buckberg GD. Myocardial protection: Entering the new millennium. J Card Surg 2002;17:447-50.
5. Breisblatt WM, Stein KL, Wolfe CJ, Follanshee WP, Capozzi J, Armitage JM, *et al*. Acute myocardial dysfunction and recovery: A common occurence after coronary bypass surgery. J Am Coll Cardiol 1990;15:1261-9.
6. Toller WG, Kresten JR, Pagel PS, Hettrick DA, Warltier DC. Sevoflurane reduces myocardial infarct size and decreases the time threshold for ischemic preconditioning in dogs. Anesthesiology 1999;91:437-46.
7. Takahata O, Ichihara K, Ogawa H. Effects of sevoflurane on ischemic myocardium in dogs. Acta Anaesthesiol Scand 1995;39:449-56.
8. Myrphy PG, Myers DS, Davies MJ, Webster NR, Jones JG. The antioxidant potential of propofol (2,6 diisopropylphenol). Br J Anaesth 1992;68:613-8.
9. Kokita N, Hara A. Propofol attenuates hydrogen peroxide induced mechanical and metabolic derangements in the isolated rat heart. Anesthesiology 1996;84:117-27.
10. Ross S, Munoz H, Piriou V, Ryder A, Foex P. A comparison of effects of fentanyl and propofol on left ventricular contractility during myocardial stunning. Acta Anaesthesiol Scand 1998;2:23-31.
11. Kalam K, Otahal P, Marwick TH. Prognostic implications of global LV dysfunction: A systematic review and meta-analysis of global longitudinal strain and ejection fraction. Heart 2014;100:1673-80.
12. Cikes M, Solomon SD. Beyond ejection fraction: An integrative approach for assessment of cardiac structure and function in heart failure. Eur Heart J 2016;37:1642–50.
13. Mirea O, Pagourelias ED, Duchenne J, Bogaert J, Thomas JD, Badano LP, *et al*. Intervendor differences in the accuracy of detecting regional functional abnormalities: A report from the EACVI-ASE Strain Standardization Task Force. JACC Cardiovasc Imaging 2018;11:25-34.
14. Sugimoto T, Dulgheru R, Bernard A, Ilardi F, Contu L, Addetia K, *et al*. Echocardiographic reference ranges for normal left ventricular 2D strain: Results from the EACVI NORRE study. Eur Heart J Cardiovasc Imaging 2017;18:833–40.
15. Amundsen BH, Helle-Valle T, Edvardsen T, Torp H, Crosby J, Lyseggen H, *et al*. Noninvasive myocardial strain measurement by speckle tracking echocardiography: Validation against sonomicrometry and tagged magnetic resonance imaging. J Am Coll Cardiol 2006;47:789-9.
16. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, *et al*.; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: A report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440-63.
17. Voigt JU, Pedrizzetti G, Lysyansky P, Marwick TH, Houle H, Baumann R, *et al*. Definition for a common standard for 2D speckle tracking echocardiography: Consensus document of the EACVI/ASE/Industry Task force to standardize deformation imaging. Eur Heart J Cardiovasc Imaging 2015;16:1-11.
18. Gaies MG, Jeffries HE, Niebler RA, Pasquali SK, Donohue JE, Yu S, *et al*. Vasoactive-Inotropic Score (VIS) is associated with outcome after infant cardiac surgery: An analysis from the pediatric cardiac critical care consortium and virtual PICU system registries. Pediatr Crit Care Med 2014;15:529-37.
19. Headrick JP, See Hoe LE, Du Toit EF, Peart JN. Opioid receptors and cardioprotection-'opioidergic conditioning' of the heart. Br J Pharmacol 2015;172:2026-50.
20. Ferferieva V, Van den Bergh A, Claus P. The relative value of strain and strain rate for defining intrinsic myocardial function. Am J Physiol Heart Circ Physiol 2012;302:188–95.
21. Marwick TH. Measurement of strain and strain rate by echocardiography: Ready for prime time? J Am Coll Cardiol 2006;47:1313–27.
22. Hashimoto I, Li X, Hejmadi Bhat A, Jones M, Zetts AD, Sahn DJ. Myocardial strain rate is a superior method for evaluation of left ventricular subendocardial function compared with tissue Doppler imaging. J Am Coll Cardiol 2003;42:1574–83.
23. Macron L, Lairez O, Nahum J. Impact of acoustic window on accuracy of longitudinal global strain: A comparison study to cardiac magnetic resonance. Eur J Echocardiogr 2011;12:394–9.
24. Dalla K, Bech-Hanssen O, Ricksten SE. General anesthesia and positive pressure ventilation suppress left and right ventricular myocardial shortening in patients without myocardial disease – A strain echocardiography study. Cardiovasc Ultrasound 2019;17:16.
25. Badran HM, Ahmed MK, Beshay MM, Zein FE. A comparative study between transthoracic and transesophageal echo modalities in evaluation of left ventricular deformation. Egypt Heart J 2019;71:4.
26. Chan J, Hanekom L, Wong C, Leano R, Cho GY, Marwick TH. Differentiation of subendocardial and transmural infarction using two-dimensional strain rate imaging to assess short-axis and long-axis myocardial function. J Am Coll Cardiol 2006;48:2026–33.
27. Roberts AJ, Spies M, Meyers SN, Moran JM, Sanders JH Jr, Lichtenthal PR, *et al*. Early and long term improvement in left ventricular performance following coronary artery bypass surgery. Surgery 1980;88:467-75.
28. Roberts AJ, Spies M, Sanders JG, Moran JM, Wilkinson CJ, Lichtenthal PR, *et al*. Serial assessment of left ventricular performance following coronary artery bypass grafting. J Thorac Cardiovasc Surg 1981;81:69-84.
29. De Hert SG, Broecke PW, Mertens E, Van Sommeren EW, De Blier IG, Stockman BA, *et al*. Sevoflurane but not propofol preserves myocardial function in coronary surgery patients. Anesthesiology 2002;97:42–9.
30. Sayin MM, Ozatamer O, Tasoz R, Kilinc K, Unal N. Propofol attenuates myocardial lipid peroxidation during coronary artery bypass grafting surgery. Br J Anaesth 2002;89:242–6.
31. Novalija E, Fujita S, Kampine JP, Stowe DF. Sevoflurane mimics ischemic preconditioning effects on coronary blood flow and nitric oxide release in isolated heart. Anesthesiology 1999;91:701–12.
32. Sutherland GR, Di Salvo G, Claus P, D'hooge J, Bijnens B. Strain and strain rate imaging: A new clinical approach to quantifying regional myocardial function. J Am Soc Echocardiogr 2004;17:788-802.
33. Yao YT, Li LH. Sevoflurane versus propofol for myocardial protection in patients undergoing coronary artery bypass grafting surgery: A meta-analysis of randomized controlled trail. Chin Med Sci J 2009;24:133-41.