

Comparison of the effects of inhalational anesthesia with desflurane and total intravenous anesthesia on cardiac biomarkers after aortic valve replacement

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ABSTRACT

Objective (s): The aim of this study was to compare the effects of using inhalational anesthesia with desflurane with that of a total intravenous (iv) anesthetic technique using midazolam-fentanyl-propofol on the release of cardiac biomarkers after aortic valve replacement (AVR) for aortic stenosis (AS). The specific objectives included (a) determination of the levels of ischemia-modified albumin (IMA) and cardiac troponin I (cTnI) as markers of myocardial injury, (b) effect on mortality, morbidity, duration of mechanical ventilation, length of Intensive Care Unit (ICU) and hospital stay, incidence of arrhythmias, pacing, cardioversion, urine output, and serum creatinine. **Methodology and Design:** Prospective randomized clinical study. **Setting:** Operation room of a cardiac surgery center of a tertiary teaching hospital. **Participants:** Seventy-six patients in New York Heart Association classification II to III presenting electively for AVR for severe symptomatic AS. **Interventions:** Patients included in the study were randomized into two groups and subjected to either a desflurane-fentanyl based technique or total IV anesthesia (TIVA). Blood samples were drawn at preordained intervals to determine the levels of IMA, cTnI, and serum creatinine. **Measurements and Main Results:** The IMA and cTnI levels were not found to be significantly different between both the study groups. Patients in the desflurane group were found to had significantly lower ICU and hospital stays and duration of postoperative mechanical ventilation as compared to those in the TIVA group. There was no difference found in mean heart rate, urine output, serum creatinine, incidence of arrhythmias, need for cardioversion, and 30-day mortality between both groups. The patients in the TIVA group had higher mean arterial pressures on weaning off cardiopulmonary bypass as well as postoperatively in the ICU and recorded lower inotrope usage. **Conclusion:** The result of our study remains ambiguous regarding the overall protective effect of desflurane in patients undergoing AVR although some benefit in terms of shorter duration of postoperative mechanical ventilation, ICU and hospital stays, as well as cTnI, were seen. However, no difference in overall outcome could be clearly established between patients who received desflurane and those that were managed solely with IV anesthetic technique using propofol.

Received: 23-08-15

Accepted: 23-09-15

Access this article online

Website: www.annals.in

PMID:
26440236

DOI:
10.4103/0971-9784.166455

Quick Response Code:



Key words: Aortic stenosis; Cardiac biomarkers; Cardiopulmonary bypass; Desflurane; Ischemia-modified albumin; Total intravenous anesthesia; Troponin I

INTRODUCTION

The protective effects of inhalational anesthetics in the prevention of ischemia-reperfusion-induced myocardial injury have been well-established in patients undergoing cardiac surgery.^[1-9,11] Most of these trials that have evaluated the efficacy of the

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Cite this article as: Kapoor PM, Taneja S, Kiran U, Rajashekhar P. Comparison of the effects of inhalational anesthesia with desflurane and total intravenous anesthesia on cardiac biomarkers after aortic valve replacement. *Ann Card Anaesth* 2015;18:502-9.

volatile anesthetic induced myocardial protection have been conducted on patients undergoing coronary bypass grafting for coronary artery disease.^[11] However, there is a paucity of published literature comparing the effects of volatile anesthetic agents with total intravenous (iv) anesthetic technique in patients undergoing valvular heart surgery. This study compares the effects of desflurane-based anesthetic regime with a total iv technique utilizing propofol, fentanyl, and midazolam on outcomes in patients undergoing aortic valve replacement (AVR) surgery. A number of studies done on patients undergoing cardiac surgery have used troponin I (cTnI) as a biomarker for myocardial injury. In this study, ischemia-modified albumin (IMA) and cTnI have been used to compare the effects of a primarily volatile based anesthetic technique with total IV anesthesia (TIVA) in a selected group of patients that were included in this study.^[1,5,9,12]

IMA is a newer marker that has gained acceptance in the evaluation of myocardial injury in recent times.^[13]

It was hypothesized that an inhalational anesthetic technique using desflurane-fentanyl would be accompanied by a more marked cardioprotective effect when compared with a TIVA technique using midazolam-fentanyl-propofol in patients undergoing AVR under cardiopulmonary bypass (CPB).

AIM AND OBJECTIVES

The primary aim of this study is to compare the effect of using desflurane-fentanyl based anesthesia with that of an iv anesthetic technique on the release of cardiac biomarkers after AVR for aortic stenosis (AS).

The specific objectives included:

- Determination of the levels of IMA and cTnI as markers of myocardial injury
- Effect on mortality, morbidity, duration of mechanical ventilation, Intensive Care Unit (ICU) and hospital length of stay (LOS), incidence of arrhythmias, pacing, cardioversion, and urine output and serum creatinine on patients subjected to these two anesthetic regimes.

METHODOLOGY

Study design

Prospective randomized clinical study.

Patient population

Approval of the Institutional Ethics Committee and written informed consent from patients was obtained. Seventy-six adult patients in New York Heart Association (NYHA) classification II-III scheduled for elective AVR with CPB for AS were included in the study. Patients were randomized by the sealed envelope technique into two groups namely, the desflurane group ($n = 36$) comprising of those who received desflurane-fentanyl anesthesia and TIVA group ($n = 40$) who would be subjected to TIVA with propofol, midazolam-fentanyl.

Inclusion criteria

Adult patients with NYHA classification II-III scheduled for elective AVR with CPB for severe symptomatic AS with valve area, $<1 \text{ cm}^2$ mean transaortic gradient $>40 \text{ mm}$, maximum aortic velocity (V_{max}) $>4 \text{ m/s}$.

Exclusion criteria

Patients with mild to moderate AS, asymptomatic severe AS patients with aortic valve area $>1 \text{ cm}^2$, mean transaortic gradient $<40 \text{ mm}$, and $V_{\text{max}} < 4 \text{ m/s}$ with left ventricular ejection fraction (LVEF) $>50\%$ were excluded from the study as were those with aortic regurgitation. Other criteria for exclusion were age <18 years, aortic cross-clamp time $>150 \text{ min}$, concomitant coronary artery disease, severe left ventricular dysfunction with LVEF $<30\%$, concomitant involvement of other valves, cardiac arrhythmias, diabetes mellitus, uncontrolled hypertension, preexisting renal dysfunction peripheral vascular disease patients, permanent or temporary pacing, and patients on intra-aortic balloon pump or those presenting for emergency surgery.

Anesthesia technique

On the day of surgery, all patients received preoperative medication, with the exception of beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin II antagonists, and diuretics. Premedication consisting of intramuscular morphine in a dose of 0.1 mg/kg and intramuscular promethazine in a dose of 0.5 mg/kg was administered to all patients 30 min prior to shifting inside the operating room. All patients received general anesthesia with endotracheal intubation facilitated by iv rocuronium in a dose of 1.2 mg/kg ; neuromuscular blockade was maintained using timed boluses of vecuronium titrated to effect throughout surgery. The depth of anesthesia was monitored using bispectral index (BIS) that was kept in a range of 40–60 during the procedure. All patients were preoxygenated with

100% oxygen and following intubation subjected to volume-controlled mechanical ventilation with oxygen in the air (fraction of inspired oxygen = 0.6) and positive end-expiratory pressure of 5 cm H₂O targeted to an end-tidal carbon dioxide level of 30–35 mmHg.

Patients in the TIVA group were administered a combination of midazolam-fentanyl-propofol along with neuromuscular blockade. Anesthetic induction in these patients was effected using fentanyl (5 µg/kg) followed by etomidate (0.3 mg/kg) given intravenously. Anesthesia was maintained using incremental doses of midazolam-fentanyl administered as needed to control hemodynamic responses to surgical stimulation. The boluses of midazolam (0.05 µg/kg) and fentanyl (2–3 µg/kg) were administered intravenously and also added to the venous reservoir of the CPB pump on an hourly basis. An iv infusion of propofol was started after intubation in a dose range of 75–150 µg/kg/min for all patients in the TIVA group and titrated to mean arterial pressure (MAP) and BIS.

Induction of general anesthesia for patients in the desflurane-fentanyl group was done using iv administration of fentanyl (2–3 µg/kg) and etomidate (0.3 mg/kg) and maintained with desflurane (0.15–1 MAC end-tidal) during the entire surgical procedure and with hourly boluses of fentanyl (1–2 µg/kg) iv. Before initiation of CPB, heparin was administered into a central vein in a dosage of 400 U/kg, to maintain the kaolin-activated clotting time (ACT) above 400 s. The ACT was measured every 30 min during CPB, and after a reversal of heparin with protamine, Magnesium sulfate in the dose of 50 mg/kg was administered on CPB on aortic cross-clamp removal. After separation from CPB, protamine sulfate was infused in a dose of 1.3 mg/mg of heparin to antagonize the effects of heparin. An additional dose of protamine 0.2 mg/kg was added in cases where the ACT was than 130 s. All patients received 0.1 U/kg of platelet transfusion for hemostasis after separation from CPB.

Following surgery, the patients were transported to the cardiothoracic surgical ICU and managed by a separate team of intensivists. Adhering to a fast track protocol, standardized intensive care was implemented, and analgesia provided by iv fentanyl and paracetamol. Extubation was performed in hemodynamically stable patients after a period of gradual weaning from mechanical ventilation.

Surgery

A mid sternotomy approach for AVR performed under CPB, and cardioplegic arrest was used in all patients included in this study. The extracorporeal CPB circuit was primed with lactated ringer solution 20 ml/kg, sodium bicarbonate (7.5%) 1 ml/kg, mannitol (20%) 0.5 g/kg, and 100 U/kg of heparin. A membrane oxygenator was used to establish a nonpulsatile blood flow between 120 and 200 ml/kg/min in all patients. Del Nido cardioplegia administered in the aortic root and repeated as per institutional protocol. Patients were cooled to 32°C. Alpha-stat acid-base management was used in both groups. Perfusion pressure was maintained between 50 and 70 mmHg. Packed red blood cell were added to pump volume during CPB, to maintain a hematocrit >30% on an as required basis.

Data collection

Baseline heart rate and MAP (HR1 and MAP1) were recorded prior to the induction of anesthesia in all patients included in this study. Thereafter, HRs and MAPs were then serially recorded just prior to institution of CPB (HR2, MAP2), on weaning off CPB (HR3, MAP3), and on arrival in ICU (HR4, MAP4). The length of ICU and hospital stay and duration of mechanical ventilation were noted commencing from the time; the patient arrived in the ICU. The number of episodes of arrhythmias, attempts of cardioversion post-CPB, and hours of inotrope usage while under anesthesia and postoperatively for 24 h in the ICU was tabulated.

Blood samples were drawn from the central venous catheter immediately after induction of anesthesia (baseline, T1) and thereafter three and 6 h post-aortic clamp removal (T2 and T3) to measure the levels of IMA. CTnI was measured at baseline after induction (T1) and at 12 h (T2) postoperatively. The plasma concentrations of CTnI and IMA were determined using commercially available chemiluminescent micro-particle and cobalt immunoassays using an automated analyzer.

The 24 and 48 hourly urine output was measured starting from induction of anesthesia and continued in the ICU postoperatively. Serum creatinine was measured at baseline prior to the induction of anesthesia (T1) and at 24 h (T2) after release of aortic cross-clamp.

Data analysis

Statistical analysis of the data collected was done using SPSS 16.0 (IBM Corporation, USA) for Microsoft

Windows. Student's *t*-test and Mann–Whitney U-test were used for normally distributed continuous data and nonnormally distributed data, respectively.

Categorical variables between the two groups were tested with the Pearson's Chi-square test. Correlation analysis was done to see the relation within each group. In addition, the paired *t*-test was applied to see the change in variables separately for each group. Repeated measure analysis was performed to determine the trend in HR in the intraoperative and postoperative period. A 2-tailed $P < 0.05$ was considered statistically significant.

RESULTS

Of a total of 76 patients included in this study, 40 patients were in the desflurane group and 36 in the TIVA group. Both the groups were similar in terms of age, sex height, weight, NYHA classification, presence of hypertension baseline HR, blood pressure, BIS, and CPB and aortic cross-clamp times during the conduct of AVR [Table 1]. The mean duration of postoperative mechanical ventilation was observed to be significantly lower among patients in the desflurane group (9.00 ± 1.28 h) than in patients included in the TIVA group (14.03 ± 2.97 h) ($P < 0.001$). Patients in the desflurane group were determined to have significantly lower ICU stay as compared to those in the TIVA group. The mean duration of ICU stay of patients in the desflurane group was 3.5 ± 1.013 days and of those included in the TIVA group was 7.92 ± 1.42 days ($P = 0.03$). The mean hospital stay was also found to be significantly lower in patients in the desflurane group (7.02 ± 1.18 days) in comparison with the TIVA group (7.92 ± 1.42 days) ($P = 0.009$).

Table 1: A comparison of demographic, baseline vital parameters, CBP and aortic cross clamp times between the two groups

Parameters	Desflurane group		TIVA group		P value
	Mean	SD	Mean	SD	
Age, years	42.35	15.13	38.36	15.370	0.258
Weight, kg	42.67	7.38	47.30	12.251	0.054
Height, cm	158.02	6.93	185.39	9.854	0.312
HR, bpm	79.61	11.427	77.05	13.083	0.369
MAP, mmHg	89.24	12.49	92.42	16.46	0.349
CPB time	90.68	23.131	93.50	25.551	0.614
AOXCT	63.68	18.512	64.81	21.503	0.806

SD: Standard deviation, HR: Heart rate, bpm: Beats/minute, MAP: Mean arterial pressure, CPB: Cardiopulmonary bypass, AOXCT: Aortic cross - clamp time depicted in minutes. A paired *t*-test was used for analysis

Patients in the TIVA group recorded significantly lower inotrope use (52.86 ± 6.91 h) than those in the desflurane group (62.08 ± 6.54 h) ($P < 0.001$). The incidence of arrhythmias and 30-day mortality were found to be comparable between the two groups (<0.05) [Table 2 and Figure 1].

The mean HRs recorded at predesignated time intervals were found to be comparable between both groups [Figure 2]. Table 3 lists the mean HRs HR1, HR2, HR3, and HR4 of patients in the desflurane and TIVA groups. In both groups, the HRs HR2, HR3, and HR4 were significantly higher than at baseline ($P < 0.001$). However, no difference was elicited when the respective HRs were compared between the two groups ($P > 0.05$). The MAPs recorded at time intervals T2 and T4 were found to be significantly higher in patients included in the TIVA group (90.75 ± 5.294 and 91.22 ± 5.519 mmHg) than in the desflurane group (85.50 ± 10.727 and 86.50 ± 11.020 mmHg) ($P = 0.10$ and 0.02), whereas the MAP values at T1 and T3 were similar in both the groups ($P > 0.05$).

There was no difference noted in the 24-h and 48-h urine outputs measured in both the groups ($P = 0.97$ and 0.53). Likewise, the serum creatinine values obtained were also not different for patients in the desflurane and TIVA groups ($P = 0.13$ and 0.27 for values obtained at T1 and T2, respectively). There was no difference in between the two groups in terms of the number of defibrillatory shocks delivered to patients in the respective groups ($P = 0.27$). The incidence of postprotamine pacing was similar in both the groups ($P = 0.84$). Nine (22.5%) of the patients in the desflurane group required atrial pacing, 20 (50.0%) were subjected to ventricular pacing and 11 (27.5%)

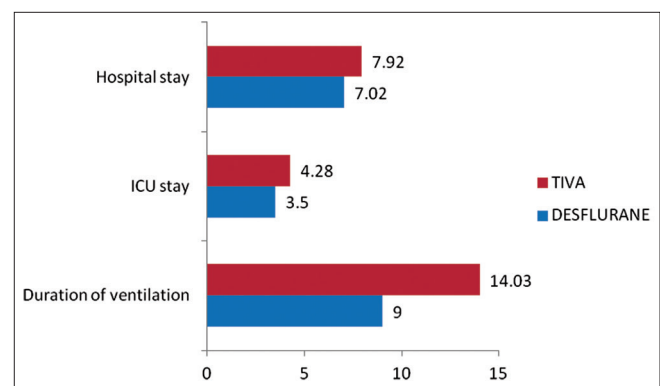


Figure 1: A representation of lengths of hospital and Intensive Care Unit stay and duration of postoperative mechanical ventilation in the desflurane and total intravenous anesthesia groups

underwent atrio-ventricular sequential pacing. The corresponding figures for patients in the TIVA group were 8 (22.2%), 16 (44.4%), and 12 (33.3%).

There was no difference in the cTnI levels obtained at various time intervals between the desflurane and TIVA groups ($P = 0.596$). The levels of cTnI obtained at T2 were significantly higher than those at T1 in the TIVA group ($P = 0.001$) as compared to the desflurane group ($P = 0.692$) [Table 4, Figure 3]. The IMA titers obtained at T1, T2, and T3 were similar for the desflurane and TIVA groups ($P = 0.78, 0.27, \text{ and } 0.166$). Within each group, the IMA levels obtained at T2 and T3 were significantly higher than baseline levels at T1 for both the groups [Table 5 and Figure 4].

DISCUSSION

In our study, the patients in the desflurane group were found to have significantly lower ICU and hospital stays and duration of postoperative mechanical ventilation as compared to those in the TIVA group [Figure 1]. There was no difference found in 30-day mortality, urine output, serum creatinine, incidence of arrhythmias, and need for cardioversion between both groups. The

patients in the TIVA group had significantly higher MAPs on weaning off CPB as well as postoperatively in ICU and had lower inotrope scores. The mean HRs of patients in the desflurane group were higher post-CPB but did not attain statistical significance [Figure 2]. The IMA and cTnI levels were not found to be significantly different between both the study groups [Figures 3 and 4]. However, the post-CPB cTnI level was significantly higher than baseline in the TIVA group, showing a cardioprotective ischemia preconditioning effect of desflurane. This is similar as found by the authors in a similar study done on cyanotic pediatric patients undergoing modified Blalock-Taussig shunt surgery.^[10]

Previously conducted trials and meta-analysis by other authors have focused on the myocardial protective effects of volatile anesthetics mainly in patients undergoing coronary artery bypass grafting procedures.^[12]

No studies are available that compare the effects of using desflurane versus TIVA in patients undergoing aortic valve surgery for AS. Among the studies published, several have compared the effects of sevoflurane and desflurane versus TIVA using propofol in patients

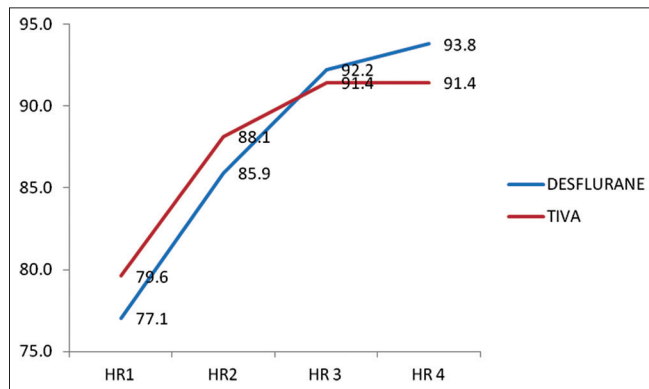


Figure 2: A comparison of mean heart rates at different time intervals between desflurane and total intravenous anesthesia groups

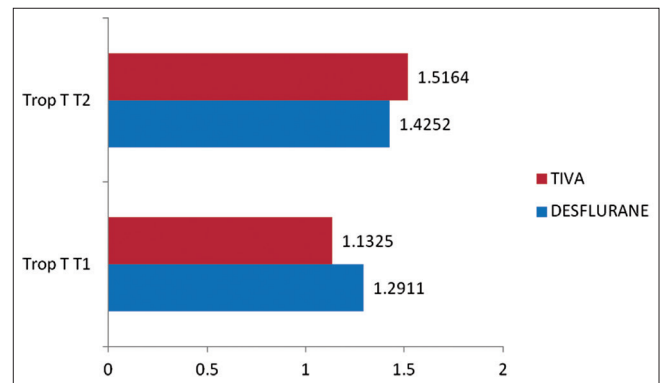


Figure 3: Cardiac troponin I levels in desflurane and total intravenous anesthesia groups

Table 2: A comparison of outcome measures between the two groups

Parameter	Mean±SD		P value	Test
	Desflurane group	TIVA group		
Duration of mechanical ventilation, hours	9±1.28	14.03±2.97	0.000	Paired t-test
ICU stay, days	3.50±1.01	4.28±1.37	0.003	Paired t-test
Hospital stay, days	7.02±1.18	7.92±1.42	0.009	Paired t-test
Duration of inotropes use, hours	62.08±6.54	52.86±6.91	0.000	Paired t-test
Incidence of SVT, percent	17.5%	16.7%	0.923	Pearson's Chi Square test
Incidence of VT/VF, percent	17.5%	19.4%	0.827	Pearson's Chi Square test
30-day mortality	3 (7.5%)	2 (5.6%)	0.733	Pearson's Chi Square test

ICU: Intensive care unit, SVT: Supraventricular tachycardia, VF: Ventricular fibrillation, SD: Standard deviation

Table 3: A comparison of heart rates recorded serially at different time intervals in the two groups

Parameters	Desflurane group		TIVA group		P value
	Mean	SD	Mean	SD	
HR1	79.61	11.427	77.05	13.083	0.369
HR2	88.14	10.393	85.90	10.157	0.346
HR3	91.44	9.996	92.22	11.310	0.752
HR4	91.43	8.204	93.80	12.654	0.342

SD: Standard deviation, TIVA: Total IV anesthesia, HR: Heart rate. HR1, HR2, HR3, HR4 denote heart rates recorded at baseline, just prior to CPB, on weaning off CPB and on arrival at ICU

Table 4: A comparison of troponin T levels between the groups

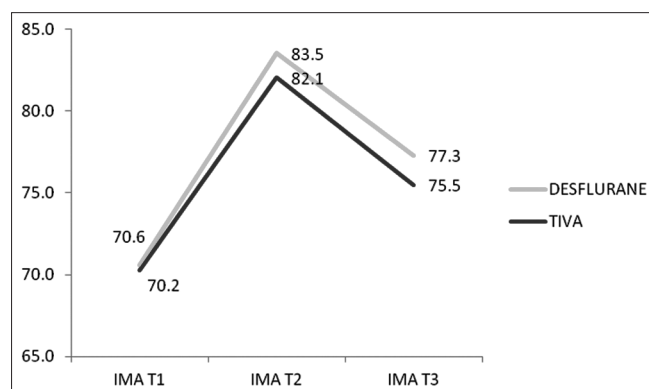
Parameters	Desflurane group		TIVA group		P value
	Mean	SD	Mean	SD	
Troponin TT1	1.2991	0.71933	1.1008	0.69938	0.596
Troponin TT2	1.4253	1.11483	1.5164	1.04356	0.257
P value	0.692		0.001		

SD: Standard deviation

Table 5: A comparison of Ischemic modifies albumin levels between the groups

Parameter	Desflurane group		TIVA group		P value
	Mean	SD	Mean	SD	
IMA1	70.583	4.8081	70.247	5.7497	0.783
IMA2	83.535	5.7233	82.050	6.0848	0.277
IMA3	77.288	5.5455	75.483	5.6936	1.4850

A t-test for was used to compare the equality of means between the two groups. SD: Standard deviation, IMA: Ischemia-modified albumin

**Figure 4: Levels of ischemia-modified albumin in desflurane and total intravenous anesthesia groups**

undergoing cardiac surgery. A significant decrease in morbidity and mortality has been shown in patients undergoing cardiac surgery who received volatile anesthetics as compared to those that were managed with TIVA using propofol in these studies.

Among the earliest trials undertaken to evaluate the efficacy of volatile anesthetics in patients undergoing coronary artery bypass graft, sevoflurane was shown to preserve hemodynamics and better preserved the myocardium as demonstrated by lower postoperative troponin levels when compared with TIVA.^[14] Several other trials have since then established the protective effects of volatile anesthetic agents on the myocardium against ischemia-reperfusion injury.^[9,15] A recently published large meta-analysis done by Landoni *et al.* has shown that the cardioprotective effects of sevoflurane and desflurane resulted in decreased perioperative mortality and morbidity in patients undergoing cardiac surgery.^[11] Sevoflurane and desflurane have both been reported to confer this cardioprotective effect.^[12,16,17,18] In addition to the lower blood gas coefficient that effects quick equilibration of alveolar-arterial concentration, desflurane is devoid of toxic metabolites such as fluorides that cause renal damage.^[15] Thus, desflurane may retain a theoretical advantage in the fast tracking of patients that undergo prolonged duration of anesthesia. This coupled with possibly even a greater degree of cardioprotection than sevoflurane could; therefore, confer an advantage on the use of desflurane as anesthetic agent for patients undergoing cardiac surgery.

Studies have shown that there is evidence that desflurane confers myocardial protection in adult patients undergoing cardiac surgery.^[9,17,18] The concern that desflurane causes sympathetically mediated tachycardia is allayed by the absence of such effects in a dose range of 0.15–1.00 MAC that is used in this study. However, a higher degree of myocardial depression seen with desflurane may offset these advantages. Although desflurane is known to exert negative inotropic effects on the myocardium,^[21] we found no difference was found in the duration of inotrope use in the two groups.

In patients undergoing mitral valve surgery with concomitant coronary artery disease, a marked decrease in postoperative cardiac troponin (cTNI) release has been demonstrated after preconditioning with desflurane.^[18] In a study by Cromheecke *et al.*,^[20] on a small group of 30 patients undergoing aortic valvular replacement, sevoflurane-based anesthesia reduced postoperative cTnI release, incidence of atrial fibrillation, and ICU stay as compared to a technique employing target controlled infusion with propofol.^[19] However, the evidence in favor of myocardial protective effects of volatile agents have been reported to be contradictory in a setting of noncoronary artery surgery.

No clear advantage was found by Landoni *et al.* in using a preconditioning protocol with volatile anesthetics in patients undergoing coronary mitral surgery.^[11]

In a study published by Kapoor Malhotra *et al.*,^[22] the effects of desflurane versus opioid anesthesia for cardiac shunt procedures in infants with cyanotic congenital heart disease were compared. Inhalational anesthesia with desflurane was proven to reduce the duration of elective ventilation, decrease ICU, and hospital LOS but without a difference in perioperative morbidity or mortality. No hemodynamic instability was encountered, and there was no evidence that desflurane exerted a negative inotropic effect. A renal protective effect was also not clearly established in patients who received desflurane. The authors expressed the need to measure cardiac and renal biomarkers to establish the protective effects of desflurane so as to further objectify their findings.

Limitations

The present study included a relatively small cohort of patients and did not rule out inter-observer bias as well as individual variations in surgical techniques between surgeons. Furthermore, some degree of myocardial protection could be attributed to fentanyl that was used in both the study groups.

CONCLUSION

The result of our study remains ambiguous regarding the overall protective effect of desflurane in patients undergoing AVR. Although some benefit in terms of shorter duration of postoperative mechanical ventilation, ICU and hospital stays as well as cTnI levels were seen. There was no difference in the biomarkers level of IMA levels between the desflurane and TIVA groups. No difference in overall outcome could be clearly established between patients who received desflurane and those that were managed solely with iv anesthetic technique using midazolam-fentanyl-propofol. It is rather difficult to make a relatively pertinent conclusion with respect to the findings from any of the two individual groups as to which is superior in AS patient. The sample size is low in the individual groups. Further, prospective multi-centered investigations are required for evaluating the additional benefits of desflurane support in the sick AS patient.

A larger cohort of patients is needed to show the benefit of either in this at risk the population of AS patients

undergoing AVR. Larger, multi-centric, and randomized controlled trials are needed to unequivocally establish as to which a better technique of the two anesthetic regimes compared in this study.

Acknowledgments

The study was undertaken at Center for Cardiovascular Sciences, All India Institute of Medical Sciences, New Delhi. The research undertaken utilized the available resources at the Institute. No funds were sought separately or sourced from any other organization.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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