ORIGINAL ARTICLE

The Comparison of Hemodynamic Stability and Arterial Oxygen Saturation During Induction of Anesthesia by Propofol versus Sevoflurane in Patients with Left Ventricular Dysfunction or Left Main Stem Stenosis Undergoing Coronary Artery Bypass Grafting

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Abstract

Objective

To compare hemodynamic changes and arterial oxygen saturation caused by Sevoflurane vs Propofol during induction of anesthesia for open heart surgery in patients with moderate to severe left ventricular dysfunction or significant left main coronary artery disease.

Methods

We conducted a retrospective study of all patients with FF < 40 % or significant left main stenosis who underwent open heart surgery from 12th of January 2016 onwards. Twenty matched consecutive patients were included in each of two groups i.e. those who underwent induction of anesthesia with Sevoflurane and those with Propofol. The demographic profiles were compared using t-test and Chi-square test for numeric and categoric variables respectively. Apart from induction of anesthesia, similar protocol for anesthetic management and monitoring were followed in both groups. The serial values of Blood Pressure (systolic, diastolic, mean), Heart Rate and peripheral Oxygen Saturation were noted at fixed times and the variations were compared using ANNOVA with repeated measures. A p-value of <0.05 was taken as significant.

Results

The changes in systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate and SpO2 within the group S and group P were significant but changes between the groups were found insignificant.

Conclusion

In our setup, both Sevoflurane and Propofol can be safely used in cardiac patients with LV dysfunction or left main coronary artery disease, under tightly controlled circumstances.

Keywords:
Cardiopulmonary bypass grafting, invasive arterial pressure, central venous pressure, left main stem coronary artery disease, minimum alveolar concentration, ejection fraction, left ventricle

Introduction

In the practice of general anesthesia, both inhalational volatile and intravenous anesthetic agents are used for the induction in patients undergoing cardiopulmonary bypass surgery. Among the inhalational anesthetic agents, sevoflurane is used for the induction because of its properties like low solubility, absence of pungency, rapid induction, maintenance of hemodynamics, excellent control over depth of anesthesia and early recovery [1]. The hemodynamic stability is required in patients with moderate to severe left ventricular dysfunction undergoing CABG. Among the inhalational agents sevoflurane is more suitable for such patients as it gradually decreases the blood pressure and heart rate, and hence fluctuations can be avoided during induction. Regarding the cardiovascular stability, it is associated with fewer direct negative ionotropic effects and lower incidence of arrhythmias and bradycardia than Isoflurane. It has protective effects on heart and lung tissue, low risk of nephrotoxicity and hepatotoxicity [2].

Propofol, an intravenous anesthetic agent, can be used safely in patients with LV dysfunction or having left main coronary artery disease. It has low incidence of post-operative nausea and vomiting due to its anti-emetic effect. It suppresses the pharyngeal reflexes, decreases the incidence of laryngospasm and bronchospasm. It can safely be used in patients undergoing Coronary Artery Bypass Grafting (CABG) suffering from renal diseases, hepatic disorders, asthma and patients prone to malignant hyperthermia due to its extra-hepatic metabolism.

In our study, the aim was to compare the changes in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and arterial oxygen saturation caused by Sevoflurane and Propofol when used for induction of anesthesia in patients undergoing CABG.

Methods

This study was retrospective observational and was conducted in the department of anesthesia, Faisalabad Institute of Cardiology, Faisalabad from 12 Jan, 2016 to 30 Dec, 2016. The sample size depended on availability of required number of patients with EF ≤40 % and LMS disease fulfilling inclusion criteria.

Inclusion Criteria

(i) Both gender male and female patients.
(ii) Patients with EF ≤ 40%.
(iii) Patients with left main stem coronary artery disease.

Exclusion Criteria

(i) Sevoflurane unacceptable to patients.
(ii) Patients with history of adverse effects of sevoflurane / propofol.
(iii) Patients with history of failed intubation during general anesthesia.
(iv) Hypotension; Systolic blood pressure <80 mmHg.
(v) Patients with history of malignant hyperthermia.
(vi) Patients with cardiogenic shock.
(vii) Patients with history of lung surgery.
(viii) Patients with Redo CABG.

Operational Definitions

Hemodynamic changes were defined as changes in systolic blood pressure, diastolic blood pressure, mean arterial pressure, and heart rate more than 30 % of baseline. Variation in SpO2 was defined as peripheral capillary oxygen saturation more than 30% of baseline. The high risk patient was defined as ant patient with EF ≤ 40% or having left main stem coronary artery stenosis of 50% or more.

Technique of Induction

All patients in pre-operative area were given pre-medication tablet midazolam 7.5 mg PO at mid-night, advised nil per oral by the nursing staff and necessary cardiac medicines with one sip of water were given at 6 am. Supplemental oxygen was started with transparent plastic face mask @ 8 L/min in pre-op area. Peripheral venous access was established with 14 or 16 gauge cannula. Monitoring of vitals like electrocardiogram (ECG), heart rate, arterial blood pressure, arterial oxygen saturation (SpO2) was started. On the day of surgery, patient was shifted from pre-operation area to operation room along with anesthesia doctor and nurse after correct identification.

During shifting, supplemental oxygen and monitoring was continued. Re-evaluation was

done on operation table. Heart rate, non-invasive blood pressure, and SpO2 monitoring with continuous ECG display of leads II &V5 with ST segment trend analysis was done. Arterial cannula of 18 or 20 gauge was inserted for invasive blood pressure (IBP) monitoring. Multi-lumen central venous catheter 7 Fr. through internal jugular vein was inserted before or after induction according to the clinical situation of patient and temperature monitoring through nasopharyngeal probe was started after induction of anesthesia. A Defibrillator, external pace maker generator, and the cardiopulmonary bypass pump were kept ready in the event that hemodynamic emergency occurs. Typed and cross-matched packed red blood cells were arranged. Emergency drugs including inotropes, vasopressors, vasodilators, atropine, calcium chloride, lidocaine, amiodarone, and heparin were prepared.

Induction was started in the presence of surgeon. A systemic gradual induction was done with continuous assessment of the degree of cardiovascular condition and anesthetic depth to minimize the risk of hemodynamic changes. In both groups, patients were given Inj. Lidocaine 1 - 2mg/kg I/V, Inj. Morphine 0.1 – 0.15mg/kg I/V and Inj. Midazolam 0.1 - 0.4 mg/kg I/V. Group S patients received 2% sevoflurane in 100% oxygen initially for 30 seconds. The inspired concentration of sevoflurane was increased to 7% until loss of consciousness and then reduced back to 2%. Group P patients received Inj. Propofol 1.25 mg/kg. After sedation, Inj. Atracurium 0.45 mg/kg I/V was used to facilitate the insertion of endotracheal tube and started manual ventilation. After achieving the complete effects of muscle relaxant drugs laryngoscopy was performed, endotracheal tube placed, checked and secured. Mechanical ventilation was started.

Maintenance of Anesthesia:

During the procedure, maintenance of anesthesia in the period before and after cardiopulmonary bypass was performed by positive pressure ventilation with 50% oxygen, 50% air, 0.5 – 2% sevoflurane, and incremental doses of muscle relaxant and opioids. During the surgery, continuous measurement and management of vitals (Systolic and diastolic blood pressure, mean arterial pressure, heart rate, SpO2, temperature), electrocardiogram lead II and V, Central Venous Pressure, end-tidal CO2, inspired oxygen concentration, tidal volume, minute ventilation, peak airway pressure, plateau airway pressure was done. Arterial blood gases, hemoglobin, blood glucose, serum electrolytes and urine output were also monitored. Blood typing, cross-matching and arrangement of blood contents especially pack cells and platelets were done. After off-bypass, required inotropic support (adrenaline, nor-adrenaline, dopamine, dobutamine), isoket (isosorbide dinitrate) and potassium chloride infusion by syringe pump was started.

Data collection:

For both groups, group S and group P, variables were recorded and analyzed which included heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and SpO2. Duration of induction was taken from the start of I/V propofol / inhalation of sevoflurane up to intubation.

a. At shifting of patient in operation room (taken as baseline data).
b. After induction of anesthesia.
c. After intubation
d. After surgical incision.

Data of each patient was entered in the prescribed pro forma and then shifted to separate table for group S and group P. Later, it was analyzed by computer software.

Statistical analysis:

ANOVA was used to analyze and compare the clinical data of both groups. Continuous variables were analyzed using t-test depending on data distribution. Categorical variables analyzed with contingency table analysis. Fisher's exact test was used to calculate the p-value and $p < 0.05$ is significa

Results:

The Table 1 compares the patient profile in both Sevoflurane and Propofol groups. It is obvious that both groups were similar in profile with p-values > 0.05 for age, gender distribution, number of bypass grafts and ASA class. However, the mean body weight was higher in Sevoflurane group compared with Propofol group by nearly 7.0 kg. Nevertheless this finding has little clinical significance.

The Table 2 summarizes the changes in blood pressure, heart rate and arterial oxygen saturation at four predefined points of time as mentioned in

Table 1
The Comparison of Patients Profile
Sevoflurane vs Propofol Group

Variables	Sevoflurane Group	Propofol Group	P
Age in Years (Mean±SD)	**52.04±8.70**	**55.56±8.98**	0.08
Weight in Kg (Mean±SD)	**77.88±11.56**	**70.44±11.10**	0.01
Ejection Fraction in % (Mean±SD)	41.52	42.2	0.37
Gender			0.21
Male n(%)	23	20	
Female n(%)	2	5	
Number of Grafts			0.99
Single n(%)	1	1	
Double n(%)	3	4	
Tripple n(%)	21	20	
ASA Class			0.50
Class III n(%)	16	17	
Class IV n(%)	9	8	
Duration of Induction in Minutes (Mean±SD)	**9.44±3.00**	**9.69±1.91**	0.37

previous section. It is obvious that there were significant changes in these parameters with p-values of <0.0001 measured by ANOVA with repeated measures. Similar, findings were noted in the Propofol group as summarized in Table 3.

The Figures 1 to 3 illustrate the comparison of above mentioned trends. It is again clear that both groups had significant variations within the groups but these changes followed similar pattern when groups were compared with each other. The p-values for the differences between the groups were all non-significant.

DISCUSSION:

The purpose of study was to seek a safe induction agent having stability of hemodynamic parameters and arterial oxygen saturation in patients with moderate to severe left ventricular dysfunction (patients with EF $\leq$ 40%) or left main stem coronary artery disease. For this purpose we selected Sevoflurane, a volatile anesthetic agent having the quality of low blood gas coefficient, pleasant non-irritant odor, rapid induction and recovery, hemodynamic stability and least changes in body organ systems as compared to other inhalational agents. Regarding the cardiovascular stability, it is associated with fewer direct negative ionotropic effects, and low

Table 2
The Effect of Anesthetic Induction with Sevoflurane
on the Hemodynamics & Oxygen Saturation

Variable	Timing	Mean± Standard Deviation	ANOVA
Systolic Pressure (n=25)	Pre Induction	134.24±9.44	SS*=11451.28 DF**=3 MS†=3817.09 F††=43.63 P=0.0001
	Pre Intubation	105.08±13.06	
	Post Induction	126.4±11.81	
	Post Incision	120.04±11.71	
Diastolic Pressure (n=25)	Pre Induction	72.64±9.48	SS=4522.36 DF=3 MS=1507.45 F=22.59 P=0.0001
	Pre Intubation	59.2±14.02	
	Post Induction	77.36±8.73	
	Post Incision	71.84±9.76	
Mean Pressure (n=25)	Pre Induction	95.48±6.84	SS=7450.43 DF=3 MS=2483.48 F=36.75 P=0.0001
	Pre Intubation	74.36±12.93	
	Post Induction	95.52±10.34	
	Post Incision	88.12±11.30	
Heart Rate (n=25)	Pre Induction	73.12±15.69	SS=3457.55 DF=3 MS=1152.52 F=7.55 P=0.0001
	Pre Intubation	70.68±15.04	
	Post Induction	84.64±15.69	
	Post Incision	82.16±10.54	
SPO2 (n=25)	Pre Induction	97.48±2.12	SS=50.8 DF=3 MS=16.93 F=21.32 P=0.0001
	Pre Intubation	98.36±1.89	
	Post Induction	98.8±2.24	
	Post Incision	99.44±1.16	

Table 3
The Effect of Anesthetic Induction with Propofol
on the Hemodynamics & Oxygen Saturation

Variable	Timing	Mean ± Standard Deviation	ANOVA
Systolic Pressure (n=25)	Pre Induction	139.05±16.33	SS=11017.27 DF=3 MS=3672.42 F=26.12 P<0.0001
	Pre Intubation	107.43±13.41	
	Post Induction	127.24±9.19	
	Post Incision	120.24±13.81	
Diastolic Pressure (n=25)	Pre Induction	72.64±12.23	SS=3249.81 DF=3 MS=1083.27 F=15.37 P=0.0001
	Pre Intubation	60.67±6.06	
	Post Induction	76.28±10.41	
	Post Incision	75.19±13.30	
Mean Pressure (n=25)	Pre Induction	94.67±16.18	SS=3978.70 DF=3 MS=1326.23 F=8.31 P=0.000104
	Pre Intubation	77.85±9.11	
	Post Induction	94.76±14.18	
	Post Incision	88.92±15.17	
Heart Rate (n=25)	Pre Induction	81.14±19.24	SS=2506.67 DF=3 MS=835.56 F=7.55 P=0.000229
	Pre Intubation	74.09±14.54	
	Post Induction	87.62±16.72	
	Post Incision	87.05±17.73	
SPO2 (n=25)	Pre Induction	97.38±1.20	SS=60.89 DF=3 MS=20.30 F=38.23 P<0.0001
	Pre Intubation	98.23±1.26	
	Post Induction	99.09±0.77	
	Post Incision	99.62±0.50	

Figure 1
Comparison of Serial Changes in Blood Pressure while Induction of Anesthesia with Sevoflurane and Propofol

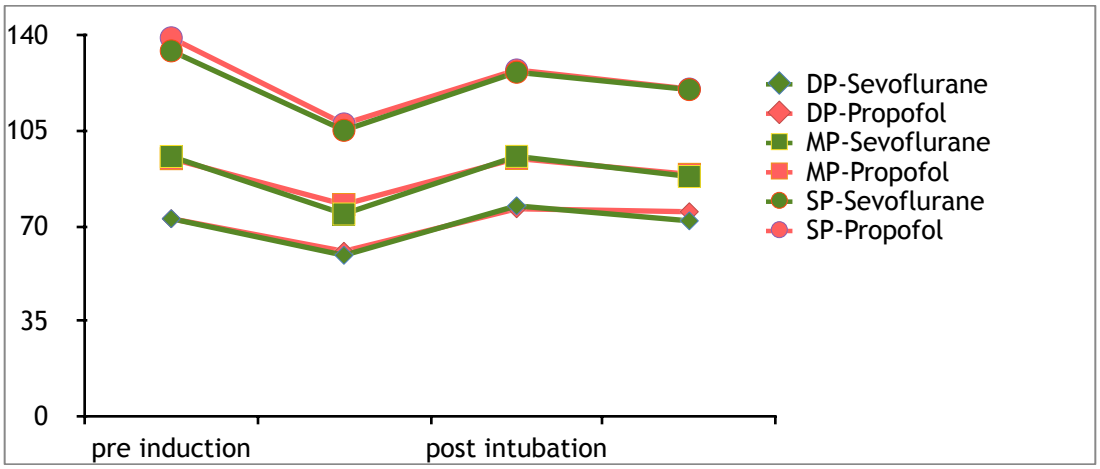


Figure 2
Comparison of Serial Changes in Heart Rate while Induction of Anesthesia with Sevoflurane and Propofol

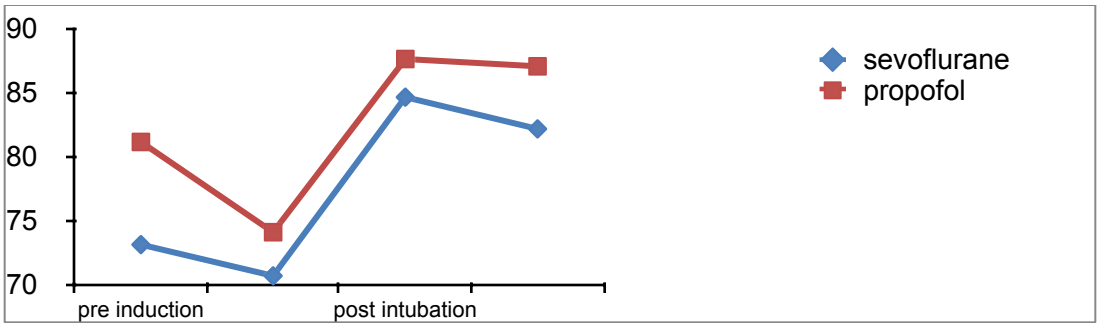
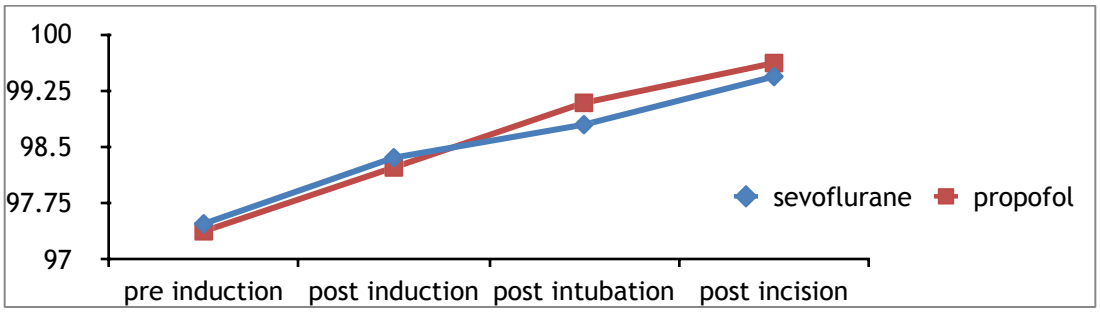


Figure 3
Comparison of Serial Changes in Oxygen Saturation while Induction of Anesthesia with Sevoflurane and Propofol



incidence of arrhythmias and bradycardia. Frequency of change in heart rate is noticed the least while maintaining a minimum alveolar concentration (MAC) level of one and half [3,4]. Heart rate remains unchanged up to 1.5 times of MAC especially in infants and children [5]. Sevoflurane is a good choice for all types of cardiac procedures like coronary artery bypass surgery, because it maintains structural and functional properties of myocardium and minimizes the risk of nephrotoxicity and hepatotoxity [6]. Sevoflurane attenuates ischemic-reperfusion injury during and after cardioplegia which is the reason of structural and functional preservation of myocardium. It also protects the heart against ischemic-reperfusion injury indirectly affecting the supply versus demand ratio, collateral flow, and direct protection through preconditioning during reperfusion. Post operatively biochemical markers of hepatic dysfunction were lower with sevoflurane based anesthesia regimen in patients undergoing coronary artery surgery with cardiopulmonary bypass [7-10]. Sevoflurane has got popularity as inhalational induction agent over other agents due to its excellent control over depth of anesthesia and hemodynamics [11]. Keeping in view the qualities of sevoflurane in comparison to other available volatile inhalational agents, we use sevoflurane as one of agents for induction of anesthesia in our practice.

Commonly available intravenous induction agents are thiopental sodium, propofol, etomidate and ketamine. Propofol is considered superior to other agents due to rapid onset, anti-emetic effect, better suppression of pharyngeal reflexes, less incidence of bronchospasm and suppression of presser response when laryngoscopy is performed for intubation. Its use is safer in patients with LV dysfunction with concomitant illness like hepatic disorders, renal failure, porphyria, asthma and in patients prone to malignant hyperthermia. It has least hangover effect after recovery. Propofol acts through activation of gamma amino butyric acid receptors-2α (GABA 2α) and is principally an inhibitory transmitter which activates ligand-gated chloride channels present on post synaptic membrane in the nociceptive pathway. The hypertensive response mediated by somato-sympathetic reflex is due to inhibition of cellular activities in caudal part of spinal trigeminal nucleus [12]. Propofol binds at distinct sites and increases GABA channels opening and conductance of chloride ions. At low concentration it increases GABA affinity and reduces GABA receptor desensitization. The sedative and hypnotic properties observed with low dose of propofol results from increase in GABA affinity. Regarding stability in cardiovascular status, propofol principally inhibits GABA mediated activity in rostral ventrolateral medulla. Moving towards the cardiovascular system, the effects of propofol to decrease mean arterial pressure, pulmonary artery pressure and reduction in heart rate results in improvement of right ventricular ejection fraction which makes its use advantageous. Propofol is believed to cause a reduction in systolic and diastolic arterial pressures by lowering systemic vascular resistance and afterload [13,14,15]. Thus among the intravenous anesthetic agents propofol is best for induction of anesthesia in patients undergoing CABG. The hemodynamic changes during induction with propofol depend on the speed of its administration and underlying cardiovascular status.

Sevoflurane had no effect on left ventricular relaxation, while neither Sevoflurane nor Propofol cause a clinical diastolic dysfunction [16]. It was established that although the mean arterial pressure values were similar after induction with Sevoflurane or Propofol, the use of sevoflurane was associated with consistently lower heart rate [17].

In this study, induction of anesthesia was done either with Sevoflurane or Propofol. Hemodynamic parameters and arterial oxygen saturations were measured before and after induction of anesthesia, after intubation and soon after surgical incision. After induction of anesthesia, hemodynamic values in both groups changed by approximately 30 % but remained within the normal range. Our study showed that patients anesthetized with sevoflurane were hemodynamically same as patients who were given propofol. It shows that hemodynamic changes in both groups were comparable but with mild advantages in heart rate and arterial oxygen saturation in Sevoflurane group. It was found that induction of anesthesia with Sevoflurane was slower and took nearly double the time as compared to Propofol. Moreover, it was also noted that few patients required atropine and nor-adrenaline to treat bradycardia and hypotension respectively in Sevoflurane group as compared to Propofol group.

The value of this study lies in the fact that we compared the effects of these induction agents in relatively high risk patients where maintaining hemodynamic stability is of paramount importance. Further studies are needed by continuous monitoring of more invasive parameters during induction of anesthesia like cardiac output, cardiac index (CI), systemic vascular resistance, pulmonary artery pressure and pulmonary vascular resistance.

Conclusion:

In our setup at FIC, both Sevoflurane or Propofol can be used safely in high risk cardiac patients with EF≤ 40% or significant LMS disease. However, it requires tightly controlled monitoring of hemodynamic parameters and prompt response in terms of pharmacological intervention, if required.

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